



# Physical Diagnosis





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*A Textbook of Symptoms and Physical Signs*

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# 1 | The History

## INTRODUCTION

THE physician's prime duty, when consulted by a patient for his ailment or disorder, is diagnosis of the latter, for without proper diagnosis, there can be no treatment or alleviation of suffering. The word diagnosis literally means "understanding through knowledge". It depends on the collection and appraisal of factual data pertaining to the case by the twin processes of (1) interrogation, history-taking, or anamnesis on the one hand, and (2) physical examination on the other.

Factual data about disease, obtained by interrogation and examination, fall into two major groups: (1) Symptoms or subjective sensations experienced by the patient, being subjective, they have to be obtained or gathered by interrogation of the patient. (2) Physical signs or objective manifestations of disease, involving alterations of structure or function, these are discovered during examination of the patient, by the physician with his unaided senses and without recourse to instruments, with the exception of the stethoscope which is permitted in the routine elicitation of physical signs.

Resort may then be had to special investigations or instrumental methods of examination, involving the use of laboratory techniques and special apparatus, in order to confirm or establish the diagnostic impression gained from a study of the symptoms and signs.

**Steps in diagnosis.** The investigation of a case with a view to clinical diagnosis should proceed systematically along the following lines:

(1) The *history*, involving the elicitation and documentation of the subjective manifestations of disease, as well as of any further information relating to the family or past history which may have a bearing on the case.

(2) *Physical examination* of the patient, involving the elicitation and recording of the physical signs of the disease.

(3) From the mass of data so collected, to reject the irrelevant or unimportant and *select* the *pertinent* or *important* data for further correlation or application.

(4) A careful *analysis* of the selected data with a view to arriving at some tentative conclusion about the diagnosis. A proper interpretation of the data will depend to a great extent on the knowledge, reasoning power and experience of the observer and on the accuracy and completeness of the data. Such an analysis will aid the examiner in carrying out further investigations with discrimination, thus saving time, expense and discomfort to the patient.

(5) Any special *investigations*, considered necessary for a final diagnosis of the disease, are carried out. These accessory aids to diagnosis must be used to supplement and not replace the time-honoured methods of interrogation and physical examination. Although investigation of a case has to be thorough, it need not be unnecessarily tedious, uneconomical or injurious to the patient. In the case of certain symptoms such as unexplained fever or loss of weight, investigation may have to be intensive or elaborate.

## HISTORY-TAKING

The *history* is a record or "recitation" of the patient's symptoms. In diagnosis, the medical history is all-important, frequently surpassing in its diagnostic importance even a thorough physical examination.

History-taking is an art with which some seem to be naturally endowed, with determination and practice, however, it can be mastered by all. The ability to elicit a good history comes with years of experience and knowledge, it cannot be mastered in the class-room. Good history-taking necessitates patience, understanding, tact, sympathy, amiability, knowledge, experience, ratiocination (reasoning) and thoroughness on the part of the interrogator.

The importance of a good history lies in the following: (1) It frequently affords a lead in the right direction or clue to diagnosis. Thus, elicitation of the various characteristics of a pain may suggest its true nature as one of angina pectoris, renal colic, acute appendicitis or duodenal ulcer. (2) It rules out or eliminates certain diagnostic possibilities. (3) It suggests further avenues of investigation. (4) It helps to focus the observer's attention to the system or systems involved. (5) Since symptoms usually precede signs, the history may afford earlier proof of disease than a clinical examination.

**Rules to observe.** In order to be useful, the history must fulfil certain criteria. The following rules must be assiduously observed during interrogation of the patient. The history, in view of its importance, must be obtained by the physician himself and not left to an assistant or secretary. It should be a confidential *tete-a-tete* between the doctor and the patient, in the presence of others, the patient may become reticent and withhold information of value. The patient should be encouraged to recite his story in his own unhurried way, without interference or interruption, unless of course he rambles on or digresses from the issue at hand, garrulous or loquacious patients have to be tactfully reminded at times of the subject under considera-

tion Leading questions, which are likely to jeopardize the value of the history by prejudicing the mind of the patient, are best avoided. The patient's own expressions and descriptions of symptoms are recorded whenever possible, provided they are intelligible. Irrelevant and rambling statements are best omitted from the written history. Any diagnosis, explanation or deduction regarding symptoms offered by the patient is best not accepted without verification. Vague and unscientific terms, such as "kidney pain", "stomach ache" and "dizzy spells", frequently employed by patients, must be carefully analyzed by questioning before recording. A rigid or stereotyped routine in questioning is not advisable, some degree of elasticity in interrogation is not only permissible but recommended. The documentation of facts must not be erratic and disjointed but proceed in logical sequence. Every symptom must be thoroughly analyzed (regarding onset, duration, character, severity, behaviour, etc.) by painstaking interrogation, it may disclose information of diagnostic value. The absence of an expected symptom, such as sweating in a case of severe precordial pain or of vomiting in a case of renal colic, may be quite significant or important and should be noted with care.

The history must be accurate, truthful and unbiased. Direct questioning, cross-questioning or special interrogation may prove necessary at times, either to elicit symptoms deliberately or unintentionally omitted by the patient, or to acquire information about the family history or personal habits of the patient. This is particularly so with nervous or reticent patients, who may require diplomatic handling and encouragement. A good history must be concise and complete. It is advisable to eliminate all redundant or unimportant data, suppress or minimize the less important, and emphasize or elaborate on the important or relevant data. A wrong or aggressive approach on the part of the interrogator may result in the patient becoming non-co-operative or cursory in his statements. Neurotic patients are prone to exaggerate symptoms and resort to the frequent use of superlatives or adjectives such as "unbearable" or "excruciating" and may recite their stories with a great show of emotion or amidst tears. They should receive a sympathetic and patient hearing but their descriptions may be suitably modified before documentation. Keen and unobtrusive observation of the patient must begin with the first contact or interview. The personality and emotional make-up of the individual, whether oversensitive, apprehensive, embarrassed, undependable or placid, must be carefully noted. Selective or discriminative questioning of the patient is only justified after years of experience of history-taking.

#### TIPS IN HISTORY-TAKING

The history of a case is usually composed of the following items (1) *vital or preliminary* data about the patient, such as name, age, sex, race, occupation, address and marital status, (2) nature and duration of the *chief complaint*



#### 4 THE HISTORY

or presenting symptom , (3) *family history* , (4) *past history* or history of past illnesses, injuries, operations, etc , (5) *personal history*, dealing mainly with personal habits, marital history and occupational history , (6) *history of present illness*, with a detailed analysis of each symptom , (7) *special* or *specific* questioning about symptoms pertaining to the system involved , and (8) a short resume of the accumulated data with the diagnostic impressions of the examiner

Regarding arrangement of these various items in the history, a slightly different order is recommended

<i>For clinical interrogation</i>	<i>For recording of facts</i>
1 Vital or preliminary data	1 Vital or preliminary data
2 Chief complaint	2. Chief complaint
3 History of present illness (origin, duration and progress)	3 Family history
4 Past history	4 Past history
5 Personal history	5 Personal history
6 Family history	6 History of present illness (origin, duration and progress)
7 Special interrogation	7 Special interrogation
8 Resume	8 Resume
9 Diagnostic impressions	9 Diagnostic impressions

During the preliminary interrogation of the patient, it is advantageous to deal with the present illness first and then proceed to elicit information about past illness and family history. In view of the natural concern of the patient over his immediate illness, questions pertaining to the state of health of family members or a review of past illnesses or episodes, at the beginning of the interview, are likely to cause annoyance or irritation and make the patient lose interest in subsequent proceedings. Also, a familiarity with the existing symptoms of the patient will simplify the subsequent task of the physician. He will be in a better position to pursue the right line of enquiry when eliciting the rest of the history.

For purposes of recording, however, the various items are best arranged in chronological order.

**Genes.** For any particular characteristic or trait, each individual possesses two factors or genes. Of a pair of genes (alleles) only one is transmitted to any particular offspring.

and it is purely a matter of chance which allele happens to be transmitted at any one time. If an individual possesses two genes which are the same he is said to be *homozygous* for that particular trait, if the two genes are different, he is *heterozygous*. A gene which is manifest in the heterozygote is *dominant*, whereas a gene which is manifest only in the homozygote is *recessive*. A trait which is determined by a gene or one of the sex chromosomes is said to be *sex-linked* and may also be either dominant or recessive, but is usually recessive, which means that the female does not show the disease except in the event of being homozygous.

It is convenient to look upon disease as a spectrum. At one end there are diseases due to infections or nutritional deficiencies or due to environmental factors (nature). At the other end there are diseases which are mostly genetic in origin (nurture). The latter group includes the so-called unifactorial and chromosomal disorders. *Unifactorial disorders* are due to a single gene defects. They are individually rare but the risk of occurrence in a family is often high, e.g. muscular dystrophy, hemophilia, Huntington's chorea. Those disorders in which there is a chromosomal abnormality include Down's syndrome, Turner's syndrome and Klinefelter's syndrome. In the middle of the spectrum are those conditions which are partly genetic and partly environmental in causation and are referred to as *multifactorial disorders*. They are comparatively common but the risk of transmission is relatively low. They include congenital malformations, hypertension, peptic ulcer and diabetes mellitus.

#### PRELIMINARY DATA

The name, age, sex, race, occupation and address of the patient are entered at the top of each history sheet.

**Age.** The age of the patient may serve to suggest certain diagnostic possibilities and rule out others. Whilst degenerative, neoplastic and vascular ailments are more common in the middle-aged or elderly, infectious fevers and congenital anomalies are commoner in childhood.

**Sex.** Certain diseases, such as cerebral haemorrhage, coronary thrombosis, haemophilia and malignancy of tongue and stomach show a special affinity for the male sex. Others, such as thyroid disorders and mammary cancer, show a similar predilection for the female sex.

**Occupation or trade** of the patient may at times afford a clue to the diagnosis. Thus lead poisoning is common in plumbers and painters, pneumoconiosis in silica workers and coal miners, and toxic jaundice in trinitrotoluene workers. Occupational asthma may result from exposure to allergenic organic dusts e.g., flour mill workers, wood workers, printers (due to sensitivity to gum acacia), workers handling furs and feathers and farmer's lung (spores of actinomycetes in mouldy hay). A high percentage of individuals with pulmonary asbestosis develop bronchial carcinoma. In leptospirosis water, soil or vegetation contaminated by the host's urine is the usual source of human infection, direct contact with hosts or their carcasses is important in occupations like abattoir workers, meat processors and milkers.

**Handedness** is important in neurological disorders.

**Residential address** of the patient may serve to focus one's attention on the various environmental factors which may be involved and may also prove useful in the subsequent follow-up of the case.

## CHIEF COMPLAINT

The first step in history-taking, after recording the preliminary data, is to state, quite briefly, the exact *nature* and *duration* of the chief complaint or presenting symptoms (e.g. "shortness of breath for two days", or "pain in the left arm for three weeks") It is better to employ expressions such as "shortness of breath" and "pain in the leg", as used by patients, rather than medical terms or equivalents such as dyspnoea or sciatica Elaboration of the presenting symptom is left to a later section of the history

In case the patient is unable to furnish the chief complaint because of loss of speech, coma, moribund condition, childhood or language difficulty, the pertinent information may be acquired from a friend or relative of the patient or deduced by observation

The exact duration of the chief complaint is of considerable importance in diagnosis For instance, the clinical significance of an abdominal pain of two hours' duration is obviously quite different from that of one of several months' or years' duration When the time factor is difficult to elicit from the patient, he may be asked to recall the last time when he was in good health

## HISTORY OF PRESENT ILLNESS

In this section, (1) the chief complaint or presenting symptom of the illness is studied in its entirety, the patient being asked to sequentially elaborate on its onset, duration, behaviour and progress A detailed analysis of the symptom at this stage will serve to narrow down the diagnostic possibilities (2) Related symptoms or attendant complaints, either volunteered by the patient or dragged out of him by tactful interrogation, are then analyzed in turn and recorded

Each symptom (e.g. abdominal pain) is taken in turn and studied from the points of view of time of onset, mode of onset, location, character, intensity, duration, behaviour, relation to the time of day, relation to meals and exertion, aggravating factors, relieving factors, and relation to associated symptoms The question of previous treatment or treatments should also be enquired into

The patient may or may not be able to provide a suitable history or background of his major and minor symptoms In the latter case, the examiner may be obliged to resort to direct or leading symptoms and narrow down the multitude of complaints to the really important or clinically significant ones The patient may require "direct" questioning about certain features, e.g., the first time he noted any illhealth or deviation from normal, the initial pattern and development of the major symptom or symptoms, its time and mode of onset and its relationship to specific events such as emotional shocks, infections, dietary indiscretions and undue physical strains

Whilst eliciting an orderly review of *other* symptoms in relation to the system affected (say the gastrointestinal tract), attention must be specially focussed on

symptoms usually related to the system affected, e.g., appetite, taste, breath, sitophobia, flatulence, belching, eructations, hiccups, pyrosis, dysphagia, nausea, vomiting, bowel habit, abdominal discomfort or pain, etc. The relationship of each symptom to the main complaint is established, e.g. Did loss of appetite precede abdominal pain? Did constipation precede or follow abdominal discomfort?

The course or behaviour of the present illness is ascertained. For instance, has the disease process been relentlessly progressive (as in case of malignancy) or episodic with remissions and relapses or stationary in its intensity and character?

### FAMILY HISTORY

The family history may yield valuable data, helpful in explaining the health problems of the patient. It affords information about the *genotype* or "inherited make-up" of the patient. The family history deals with the nature, duration and outcome of illnesses in the close relatives of the patient, the ages of living family members and the ages at death of the lost ones.

The knowledge of an illness in the family can lead to early detection and treatment as in the case of Wilson's disease where there is an excess of copper, or such diseases as diabetes mellitus. Genetic influences may be the basis of metabolic change (gout, hyperglycemia, hypercholesterolemia), brain dysfunction (Huntington's chorea, schizophrenia), or structural change in the heart (cardiomyopathy), kidney (polycystic disease), nerve and skin (Von Recklinghausen's disease), or cardiovascular disturbances (ischemic heart disease, hypertension) or familial cancers (e.g. breast) or polyposis of the colon where prophylactic colectomy may prevent the occurrence of cancer.

Within families there is a variation in the severity of the process and knowing the pattern of severity in relatives may be a guide to therapy. For instance cerebral haemorrhage in parents suffering from hypertension would lead to more aggressive approach to therapy of raised blood pressure in adults.

*Consanguinity*, in the parents must also be enquired into.

Vague terms, such as "old age", "stroke", "fits", "debility" and "heart failure", are frequently employed by patients to explain the cause of death in relatives, such terms are useless without proper amplification or elaboration.

Enquiries about the familial incidence of disease should apply to close relatives only (parents, grand-parents, brothers, sisters, aunts, uncles and cousins). The longevity or average life-span of members of the patient's family should be ascertained, whilst some families are proverbially long-lived, in others premature deaths from cerebrovascular and cardiovascular accidents are common.

In the event of a disease being described in several members of the family, a familial or hereditary incidence need not necessarily be presumed, as such an eventuality can also result from contact infection, contagiousness or common environmental influence.

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The *marital* history is best included in this section. Enquiries should be made about the health of the spouse, number of children, pregnancies, miscarriages and abortions. A series of miscarriages should make one suspect syphilitic infection. In the event of developmental anomalies in the offspring, specific enquiry is justified regarding cousin-marriages, German measles, trauma, irradiation and toxoplasmosis, factors which may have bearing on such an eventuality.

## PAST HISTORY

This includes a review of (1) past illnesses, with a pointed reference to important entities such as rheumatic fever, syphilis, epileptic fits or convulsions and hypertension, (2) injuries or traumatic lesions, (3) previous operations, (4) childhood diseases, (5) the countries of residence, certain diseases like malaria, ankylostomiasis, kala-azar and rickettsial infections being more prevalent in the tropics and sub-tropics, (6) countries visited during past travels, and (7) ingestion, application or parenteral administration of toxic or potentially toxic drugs in the past.

A review of *past illnesses* may afford information of diagnostic value. (1) The existing illness may be related to or be a sequel of some past illness such as rheumatic fever, syphilis, encephalitis, meningitis, chorea or scarlet fever, (2) history of certain viral or rickettsial infections, such as small-pox, mumps, infective hepatitis and typhus in the past, infections which confer a life-long immunity on the patient, (3) a history of bronchial asthma, gout, epilepsy, urticaria or pernicious anaemia in the past, conditions which display a tendency to relapse or recurrence.

A history of a severe trauma or fall in the past may serve to explain otherwise obscure or unexplained symptoms, such as the sciatic pain of an intervertebral disc lesion or the progressive paraplegia of spinal injury. The exact nature of a surgical operation in the past may afford a clue to the present ailment (e.g. unilateral oedema of the arm secondary to radical mastectomy in the past), or may rule out certain possibilities (e.g. pain in the right iliac fossa in a patient who has already had an appendicectomy).

In many cases a diagnosis offered by the patient for some former disease may require further enquiry in order to ascertain its validity. For example, a diagnosis of typhoid fever or influenza is often loosely employed by patients to cover a multitude of entirely different ailments such as pneumonia, rheumatic fever and endocarditis. All statements pertaining to illnesses in the past must therefore be verified by cross-questioning before acceptance.

When a past illness appears to bear a relationship to the present ailment, for instance, in a case of mitral stenosis, a direct enquiry may have to be made for a history of growing pains, aches or arthritis, frequent sore throats or streptococcal infections, tonsillectomy, chorea or muscle twitchings, attacks of nose-bleeds, or of skin rashes with joint pains in the past. There may be a history of having received aspirin, salicylates, corticosteroids or penicillin for

long periods of time in the past or of having been suspected of or treated for heart murmurs. On the other hand, unimportant childhood ailments like chicken-pox and measles need not be amplified upon.

## PERSONAL HISTORY

This is particularly important in cases of nervous, psychosomatic and nutritional disorders. The following features should receive particular attention.

(1) **Personal habits** Hours of sleep, recreation, hobbies, bowel actions, exercise, hours of work, menses, etc. The daily routine of the patient must be carefully enquired into.

(2) **Addictions** Tea, coffee, alcohol, smoking or chewing of tobacco, addiction to heroine or cocaine, and the habitual use of sleeping pills, cathartics, aphrodisiacs or stimulants. The exact quantity and nature of the offending substance must be ascertained and recorded (e.g. 20 cigarettes a day, or 2 pegs of whisky every evening). Information of this type may be useful in cases of thromboangitis obliterans, duodenal ulcer, delirium tremens, polyneuritis, lung cancer, coronary thrombosis, sudden blindness, cirrhosis of liver, vitamin deficiencies, blood dyscrasias, Korsakoff's psychosis and chronic pharyngitis.

(3) **Occupational history**. The exact nature of the patient's occupation must be ascertained. This is important because of particular hazards associated with certain occupations, e.g. wrist-drop or acute abdominal colic in plumbers or painters exposed to lead poisoning, carbon monoxide poisoning in automobile workers, pneumoconiosis in those exposed to dusts and nystagmus in miners. Past occupations must also be enquired into, as they may be responsible for the existing symptoms of the patient.

The following features are worth investigating when dealing with occupational hazards: the presence of dusts, overcrowding, lack of sanitary facilities, poor hygiene, presence of insects, gas contaminations, abnormal temperatures, defective ventilation or lighting, and hours of work and rest.

(4) **Residential history** The place of residence, whether urban or rural, whether inland, mountainous or close to the sea, and details of ventilation, sanitation, heating, drainage, cleanliness, damp, food, water supply and overcrowding should all be enquired into; the existence of cases of infectious diseases in the neighbourhood should be ascertained.

(5) **Social history** The social life, economic status, side activities, sex experiences, emotional make-up and psychological conflicts of the patient should be enquired into with tact and understanding. The social history is important in view of the unquestionable influence of emotional mal-adjustments on the causation of disease. When elicited with care, the personal history will reveal the personality of the individual and thus provide a better evaluation of the history.



## SPECIAL OR SYSTEMIC INTERROGATION

Depending on which system or systems of the body are incriminated or considered involved, on the basis of the patient's history, specific interrogation of the patient is carried out with a view to eliciting the maximum amount of information about that system. For instance, with a chief complaint of abdominal pain, suspicion would naturally fall on the alimentary system and specific questioning would be directed towards symptoms like appetite, flatulence, vomiting, eructations, constipation and diarrhoea.

## RESUME AND DIAGNOSTIC IMPRESSIONS

At the end of the history, a short summary or resume outlining the salient or important facts of the case is prepared. The diagnostic impressions or tentative conclusions of the examiner are then added. Routine adoption of such a plan tends to narrow down the subsequent field of exploration and cut out unnecessary examinations or investigations. It is important to note that a disorder or disease (such as cardiac neurosis) may at times be unintentionally or inadvertently provoked either by autosuggestion or by a stray remark from the physician (iatrogenic or iatric disorder), or through some faulty technical procedure or treatment (iatrotechnical disorder).

# 2 | Symptomatology

SUBJECTIVE manifestations or phenomena complained of or experienced by patients, in association with disease-processes are referred to as symptoms. They are of great diagnostic value, when properly elucidated, interpreted and analyzed and may, at times, suggest the right diagnosis even before physical examination is carried out.

A study of symptoms is an integral and preliminary part of any clinical examination. The science of symptoms is called symptomatology. A group or ensemble of symptoms, characteristic of a disease-process, is referred to as a *symptom-complex*. By *pathognomonic symptom* is meant a symptom characteristic enough to clinch the right diagnosis.

It is important to realize, that a symptom, although strongly suggestive of disease of one system, may also be encountered in diseases affecting one or more of the other systems. For instance, dyspnoea, although suggestive of heart disease, may also be noted in respiratory and renal diseases. Similarly, fits or convulsions, although usually neurological in origin, may be due to heart disease (Stokes-Adams attack) or renal failure.

## CLASSIFICATION OF IMPORTANT SYMPTOMS

### 1 Cardiovascular

Dyspnoea  
Precordial pain  
Palpitation  
Oedema  
Syncope

### 2 Respiratory

Cough  
Expectoration  
Chest pain  
Dyspnoea  
Haemoptysis  
Hoarseness of voice  
Stridor

### 3 Gastrointestinal

Abdominal pain  
Anorexia  
Vomiting and Nausea  
Haematemesis and Melaena  
Flatulence (gas)  
Jaundice

### 4 Neurological

Fits or convulsions  
Vertigo  
Headache  
Involuntary movements  
Coma  
Disturbance of vision

Constipation  
Diarrhoea  
Dysphagia  
Hiccough  
Abdominal distension

#### 5 Genitourinary

Urinary disturbances  
Haematuria  
Impotence

Disorder of speech  
Motor weakness  
Tinnitus  
Deafness  
Pain in the upper limb  
Pain in the lower limb  
Sensory symptoms

#### 6 Miscellaneous

Fever  
Pallor  
Loss of weight  
Backache  
Pigmentation

The majority of symptoms mentioned above are considered in alphabetical order in this chapter. The remaining symptoms are incorporated in appropriate chapters dealing with the various symptoms.

## STUDY OF INDIVIDUAL SYMPTOMS

### ABDOMINAL PAIN

Abdominal pain, like pain in any other part of the body, may be looked upon as a safety device or beneficial manifestation on the part of Nature, as it warns the sufferer of something being wrong.

Pain may be of several varieties, viz (1) *superficial* pain, which is felt in the cutaneous structures, (2) *deep* pain in the bones, muscles and blood vessels, (3) *visceral* pain, due to distension, ischaemia or obstruction of viscus, it is heavy, dull, spasmodic and deeply situated, (4) *referred* pain, although arising in a deep-seated point is felt superficially in the cutaneous structures, it is continuous, superficial, localized and associated with tenderness, (5) *conditioned* pain arises on movement and after trauma (long after the trauma has healed) (6) *psychogenic* pain, which has no physical or organic basis, and depends on the mental sphere.

Abdominal pain may be of parietal origin, due to irritation of spinal nerves, true abdominal visceral pain, referred pain or somatic pain. Many sensations, not ordinarily described as pain, may arise from the same type of stimulus. Thus burning, gnawing or vague fullness may each be initiated by the same stimulus that is capable of producing pain.

#### CAUSES

- (1) *Causes within the abdominal wall* Neuralgia, fibrositis, herpes zoster
- (2) *Intra-abdominal causes* (a) *Visceral* Intestinal, biliary or renal colic, ulcer, neoplasm or inflammation of gastrointestinal tract, liver, gall-bladder, kidney or ovary, mesenteric artery thrombosis (b) *Peritoneal* Inflammation of peritoneum, or perforative peritonitis, peritoneal deposits

(3) *Extra-abdominal causes* Coronary occlusion, pneumonia, diaphragmatic pleurisy, referred pain from spine

#### METHOD OF INQUIRY

(1) *Nature of onset* : Gallstone or renal colic may start suddenly without previous indication of disease Pain of peptic ulcer is referred to as distress or discomfort weeks before the actual pain starts

(2) *Site of pain or discomfort* :

(a) *Epigastric* Peptic ulcer, acute appendicitis (during the first few hours), acute cholecystitis, mild inflammatory lesions of small intestine, functional spasm Mid-epigastric discomfort may be due to disease of liver, bile ducts or pancreas

(b) *Umbilical* (or peri-umbilical) Functional or organic disease of small intestine

(c) *Upper right quadrant* . Disease of liver, gall-bladder or biliary passage

(d) *Upper left quadrant* Pressure type of pain or discomfort may be due to pocketing of gas in the splenic flexure of the colon , affections of pancreas, spleen, transverse colon, splenic flexure or upper part of descending colon

(e) *Lower left quadrant* Spastic colon, colitis, diverticulitis, obstructive lesion of colon, disease of left urinary tract or left adnexa

(f) *Lower right quadrant* Diseases of appendix, terminal ileum, mesentery and caecum Also, to be taken into consideration are retroperitoneal affections, right adnexal diseases and lesions of spinal cord and vertebrae

(g) *Back pain* Common duct or ureteric calculus, lesions of kidney or pancreas, mesenteric glandular disease Involvement of posterior parietal peritoneum or mesenteric attachment of bowel may also be associated with pain in the back

(3) *Character and severity of pain*. A peptic ulcer patient usually complains of a gnawing or burning type of pain Colicky pain, due to spasm, is often agonizing in character and may be due to stone within the common bile duct or ureter At times, the pain in such cases is not intermittent or colicky, but is slight at the onset, steadily becoming more intense In intestinal spasm, pain is rhythmically intermittent, with brief periods of intense pain followed by longer intervals of remission When an actual attack of pain is not observed, the severity of the pain can be judged on the basis of analgesics required , in severe cases, complete inactivity may be caused by pain

(4) *Duration and periodicity of pain* : Pains of brief duration are, as a rule, of mechanical origin, as in renal colic Long-lasting pains are usually due to organic disease, either inflammatory or neoplastic The pain of peptic ulcer occurs in bouts, lasting for weeks or months, interspersed with varying

intervals of freedom The pain of pelvic inflammatory disease tends to increase during menstruation A constant pain, lasting for weeks or months, suggests the possibility of malignancy or a low grade inflammatory process, in contact with the peritoneum

(5) **Radiation** Pain of renal colic may be referred to the testicle or labia, and that of gall-bladder disease to the angle of the right scapula, interscapular pain may be due to chronic pancreatitis

(6) **Pain relationships** Attacks of pain may coincide with certain physiologic functions or bodily activities (a) *Relation to ingestion of food* In duodenal ulcer, pain and discomfort occur two to four hours after the ingestion of food Pain coming on within a few minutes of eating is not commonly due to an organic lesion, although it may be due to reduced stomach capacity from malignancy Since eating provokes active peristalsis, the arrival of a peristaltic wave to the zone of inflammation within the small or large bowel may initiate pain or distress Attacks of biliary colic frequently occur about three to five hours after the intake of a heavy meal (b) *Relation to physical effort* Pain after a car-ride or bus-ride on a bad road may be due to the displacement of a renal or gall-bladder calculus Adhesions or peritoneal bands may predispose towards abdominal discomfort during physical exertion or change of posture (c) *Quality of food* Fats may initiate gall-bladder attacks Roughage may produce discomfort in patients with functional or organic disorders Food idiosyncrasy is suspected when symptoms occur repeatedly after certain foods only

(7) **Relief of pain** Pain of peptic ulcer is commonly relieved by taking food or alkalis and by vomiting Pain of gallstone or ureteric colic may only respond to opiates Epigastric pain that is worse on lying down and relieved by sitting up suggests diaphragmatic hernia or pancreatitis

(8) **Associated symptoms** Vomiting is common with gallstone colic Also associated are a catch in the breath and perhaps jaundice Dysuria and haematuria are common in renal colic The association of urticaria with abdominal pain should make one think of an allergic disturbance

## AEROPHAGIA

Excessive swallowing of air into the gastrointestinal tract, independently of deglutition, is referred to as *aerophagia*, *aerophagy*, *eructatio nervosa* or as "air-swallowing"

**Mechanism** Normally, the small amount of air that is swallowed by all individuals during the act of swallowing of solids, liquids or saliva, occasions no discomfort being easily evacuated through the mouth (eructation) or expelled per rectum (passage of flatus), whenever in excess When however, excessive accumulation of swallowed air occurs at one or more gastrointestinal

sites, its evacuation or passage is attended by unpleasant subjective manifestations, the condition, no longer physiological, is referred to as *aerophagia*. Contrary to common belief, air or gas is not generated within the stomach or intestines (except perhaps in carbohydrate dyspepsia), but is swallowed through the mouth, often at unbelievable rates. Over sixty per cent of intestinal gas is swallowed air, whilst only ten per cent is attributable to intestinal fermentation of food.

#### CAUSES

(1) *Dietary habits* Aerated waters, cokes, beer, bolting of food, excess of fluids, chewing gum

(2) *Addictions* Smoking, alcohol, betel nut, pan habit

(3) *Oral causes* hyper- or hypo-salivation, xerostomia, mouth-breathing

(4) *Gastrointestinal causes* Gastritis, duodenitis, ulceration, hiatus hernia, cholecystitis, cholelithiasis, irritable colon

(5) *Psychogenic* Anxiety states, neuroses, depression, emotional disturbances

(6) *Miscellaneous* Post-nasal drip, post-operative swallowing of air, angina pectoris or myocardial infarction, pulmonary embolism

The importance of aerophagia lies in that, by provoking severe symptoms, it may either *mask* or *simulate* serious organic diseases (e.g. cholecystitis, pancreatitis, angina pectoris, myocardial infarction, peptic ulceration or gallstones). Besides *symptoms*, such as frequent belching or eructation, post-prandial fullness or distension, intestinal flatulence, borborygmi, early satiety, hiccups and passage of flatus per rectum, aerophagia may give rise to important *clinical syndromes* or functional disorders, viz. (1) Eructatio nervosa or periodic, functional, oesophageal belching, a form of exhibitionism in a confirmed neurotic. (2) Magenblase, with a massive "air-bubble" in the stomach and unpleasant side-effects. (3) Pseudo-angina or angina-like pain in the chest. (4) Gastric pneumatosis with a feeling of suffocation and shallow breathing. (5) Splenic flexure syndrome with trapping of air within an angulated splenic flexure, resulting in chest pain, palpitation, choking, apprehension and a wrong diagnosis, at times, of coronary heart disease.

#### DISORDERS OF APPETITE

By "appetite" is meant a "desire for food" or "the pleasure of eating". Disorders of appetite, which include increase, decrease or perversion of appetite, may arise from diverse causes and in many ways.

*Loss of appetite* or "loss of desire to eat" is described as *anorexia*, "lack of desire to eat" as "physiological anorexia" or *satiety* and absence of satisfaction after eating as *acoria*, and a "fear of eating" because of past or present unpleasant experiences after eating is referred to as *sitophobia* (e.g. in peptic

ulceration) *Exaggerated or excessive appetite*, with food-ingestion in excess of metabolic requirements, of organic or functional origin, is described as *polyphagia* or *hyperorexia*, and excessive hunger or urge to ingest food soon after eating (usually secondary to hysteria or obsessional psychosis), even in the absence of a desire to eat, as *bulimia* or "wolf's hunger" Excessive appetite may be encountered in diabetes mellitus or thyrotoxicosis

A *perversion of appetite* or craving for unusual or abnormal foods is described as *parorexia* This may be for accepted items of diet (e.g. salt, sugar or spice), for so-called inedible items or unusual substances (e.g. chalk, plaster, earth, leaves or hair) in which case it is referred to as *geophagia* or *pica*, or for offensive items (such as faeces or urine) as in *allotriophagia* *Perverted appetite* may be met with at times in children with helminthiasis, women during pregnancy and in the insane

*Hunger* is not synonymous with appetite, being defined a physiological state caused by deprivation of food and abolished or relieved by the ingestion of food It is associated with so-called "hunger-sensations" (e.g. epigastric pang, irritability, weakness, tiredness, trembling and dizziness) and "hunger behaviour" (e.g. excessive motor activity) Excessive craving for fluids or water is referred to as *thirst*

*Anorexia* and *thirst*, in view of their importance, are dealt with separately

## ANOREXIA

A marked decrease or absence of the sensation of hunger This symptom may be associated with a wide variety of diseases and may be relative or absolute

### CAUSES

(1) *Physiological* Inactive life, irregular eating or drinking, excessive carbohydrate consumption between meals

(2) *Infections and chronic diseases* Acute febrile illness, pulmonary tuberculosis, congestive cardiac failure, uraemia, cirrhosis of liver

(3) *Lessened metabolism* Addison's disease, myxoedema

(4) *Avitaminosis* Particularly deficiency of vitamin B-complex

(5) *Local gastric causes* Carcinoma, gastritis

(6) *Endocrinopathies* Panhypopituitarism, hyperparathyroidism

(7) *Neurosis* Depressive psychosis, anorexia nervosa

*Aversion* for certain items of food may also be noted Aversion for milk and milk products may be an early sign of alkalosis (in peptic ulcer cases kept on alkalis), whilst that for meat may be encountered in early cases of gastric carcinoma

## METHOD OF INQUIRY

- (1) *History of emotional upset or of neurosis*
- (2) *Fever* Presence of persistent fever suggests infection like tuberculosis or subacute bacterial endocarditis
- (3) *Dietary history* to suggest possibility of malnutrition and avitaminosis
- (4) *Loss of weight* Rapid loss of weight in malignancy, such as carcinoma of stomach
- (5) *History of taking drugs* (which are known to depress appetite) like amphetamine, ammonium chloride, sulphonamides
- (6) *History of chronic alcoholism* suggests chronic gastritis or cirrhosis
- (7) *Associated symptoms* Physical and mental retardation in myxoedema, pigmentation of skin in Addison's disease, dyspnoea and oedema in cardiac failure

## BACKACHE

Pain in the back or along the spinal column is a very common symptom, due to a variety of causes. The diagnostic problem is more or less the same as with pain anywhere else in the body. In the case of backache, a careful history is often of great value in diagnosis.

The pain may be mild or severe, sharp, dull or aching in character, constant or periodic, increased by rest or movement, unilateral or bilateral, localized or diffuse and affecting a solitary region (cervical, dorsal, lumbo-sacral, sacroiliac, coccygeal) or the entire spine. Low backache is particularly common in clinical practice.

## CAUSES

- (1) *Lesions of the vertebral column* Prolapsed disc, trauma, spondylitis, osteoarthritis, osteoporosis, carcinomatous deposits, Paget's disease
- (2) *Lesions of soft tissues* (ligamentous and muscular causes) Fibrositis, myositis, ruptured or strained ligaments or muscles
- (3) *Lesions of spinal cord* Spinal tumour, syphilitic myelitis
- (4) *Faulty posture* flat feet or bodily deformity
- (5) *Intrathoracic, intra-abdominal or pelvic diseases (reflex causes)* Diseases of stomach, gall-bladder, pancreas, aneurysms, kidney affections, salpingitis, ovarian tumours
- (6) *Psychogenic* Hysteria, anxiety state, neurasthenia, "railway spine"

## METHOD OF INQUIRY

- (1) *Site and character of pains* The pain is sacral or sacroiliac in pelvic diseases. The pain of prolapsed disc tends to vary, with remissions and relapses.



(2) *Mode of onset* Acute onset in lumbago and prolapsed intervertebral disc In spinal subarachnoid haemorrhage, a sudden and agonizing local pain in the back is usually accompanied by root pains and sciatica In dissecting aneurysm, the patient may complain of severe pain in chest, abdomen or groins

(3) *Distribution of pain* The pain of prolapsed disc is often localized to one side and low down over the ilio-sacral region The pain extends into the buttock and corresponds to the distribution of one or more nerve roots Pains, due to diseases of joints, ligaments and muscles have no such anatomical distributions

(4) *Duration* In disc lesions, backache usually precedes root-pain (lumbago before sciatica) and recurrent attacks of backache tend to be more and more severe Short duration and acute pain are characteristic of lumbago

(5) *Aggravating or relieving factors* Pelvic pain in women is worse during menstruation Disc pain is aggravated by movement, exertion and bending forward, but is eased by resting, particularly in some special position

(6) *History of injury, recent or remote* Fractures of the vertebrae can occur from relatively minor injuries, especially in the presence of bone disease

## CHEST PAIN

### INTRODUCTION

Pain is an unpleasant perception caused by stimulation of sensory end-organs and afferent tracts Its appreciation depends to a great extent on the "threshold" of the individual to pain Anterior thoracic pain is one of the commonest and most important symptoms of cardiovascular and respiratory diseases

### TYPES

Amongst the common sites of maximum intensity or localization of anterior thoracic pain are (1) *retrosternal* or *substernal* pain, which is frequently associated with organic heart disease, but may be extracardiac (as in mediastinal new growths), (2) *precordial* pain, over the region of the heart, (3) *left inframammary* pain, often functional in origin, and (4) *left supramammary* or upper left parasternal pain

### CLASSIFICATION OF CAUSES

A *Thoracic or chest wall pains* These are usually localized, continuous, often associated with local tenderness and are superficial or deep

(1) *Diseases of skin and subcutaneous tissues* Panniculitis or cellulitis and adiposus dolorosa (Dercum's disease)

- (2) *Affections of the mammary glands* Chronic mastitis and pre-menstrual pain
- (3) *Muscular affections* Myositis, myofibrositis, epidemic myalgia and pectoral strain
- (4) *Diseases of nerves* Herpes zoster, neuritis, neurofibromatosis, pressure on nerves and intercostal neuralgia
- (5) *Diseases of bones and joints* Costo-chondral arthritis (Tietz's syndrome), slipping rib, local rib lesions (such as metastasis and myeloma), ligamentous strain of cervico-dorsal spine, osteoarthritis of spine, rupture or prolapse of sixth cervical intervertebral disc, scalenus anticus syndrome and scapulo-humeral or shoulder-hand syndrome

## B Intrathoracic causes .

- (1) *Diseases of the heart* Angina pectoris, coronary occlusion, intermediate coronary syndrome, pericarditis, myocarditis and endocarditis
- (2) *Diseases of the aorta* Aortic aneurysm, dissecting aneurysm, aortalgia and specific aortitis
- (3) *Diseases of the respiratory system* Pleurisy, pneumo-thorax, acute pulmonary embolism, pneumonia, massive collapse, lung abscess, carcinoma of lung, tracheitis and bronchitis
- (4) *Diseases of the mediastinum* Mediastinitis, mediastinal new growth and mediastinal emphysema
- (5) *Diseases of the oesophagus* Cardiospasm, oesophageal spasm, oesophageal ulcer and oesophageal obstruction
- (6) *Diseases of the diaphragm* Acute diaphragmitis, diaphragmatic tic or flutter, diaphragmatic hiatus or para-oesophageal hernia

## C Extrathoracic causes .

- (1) *Intra-abdominal disease* Peptic ulcer, colonic distension (splenic flexure syndrome), gastrophrenic or gastroduodenal syndrome, diseases of the gall-bladder, liver abscess, subphrenic abscess and acute pancreatitis
- (2) *Neurological diseases* Tabes dorsalis, transverse myelitis, disseminated sclerosis, spinal tumour, fractures and segmental pains of radiculitis
- (3) *Endocrine causes* Menopause, premenstrual pain and pheochromocytoma
- (4) *Spinal pain* Degenerative changes in the lower cervical or upper thoracic spine may give rise to pain which is referred to the anterior chest
- (5) *Psychogenic causes* Neurocirculatory asthenia and cardiac neurosis

## METHOD OF INQUIRY

- (1) *Onset* Acute in myocardial infarction, pericarditis
- (2) *Site or location of pain* Pain midsternal or substernal in angina.
- (3) *Type of pain* Pressing, constricting, vice-like, mere heaviness or burning type of pain in angina, dull heavy feeling beneath the left breast in neurocirculatory asthenia. Pain arising from the pleura is sharp and worse on breathing or coughing. In malignant tumours of the mediastinum or in aortic aneurysm, pain is constant and of a boring quality. In dissecting aneurysm there is a severe tearing pain maximal in intensity at the onset.
- (4) *Radiation of pain* Pain of angina or of myocardial infarction may radiate to shoulders or arms or into the neck or jaws. Pain which radiates along the course of one or two intercostal nerves usually has its origin in some part of the local sensory apparatus.
- (5) *Precipitating or aggravating causes* Effort or excitement tends to precipitate pain or discomfort in angina pectoris. The "heartache" in neurocirculatory asthenia is related to fatigue rather than to effort. Pain associated with overeating and recumbency is characteristic of hiatus hernia. Oesophageal pain is aggravated by swallowing. Pain due to diseases of thoracic cage is as a rule aggravated by coughing sneezing or straining. Pain of dry pleurisy tends to be aggravated by lying on the affected side and relieved by lying on the sound side. Pain of pericarditis may be aggravated by breathing, coughing, swallowing or twisting the trunk. Myositis affecting the pectoral muscle will be aggravated by movement or contraction of the muscle.
- (6) *Relief of pain* Pain of angina can be relieved by resting or by nitroglycerin. Pain of pericarditis may be abated by leaning forward in the sitting posture.
- (7) *Duration of pain* Pain of neurocirculatory asthenia may last several hours or days and may be almost constant. Pain of angina usually lasts for few seconds.
- (8) *Associated features* Frequent sighing and palpitation are common in neurocirculatory asthenia, sweating and dyspnoea may accompany pain of myocardial infarction. Haemoptysis is common in pulmonary infarction.

## COMA

Coma may be defined as a state of prolonged unconsciousness from which the patient cannot be aroused. It may be preceded by a state of stupor or diminished consciousness. The distinction between coma and syncope is a relative one, depending on the duration of the unconscious state. Information obtained from relatives or witnesses will often provide valuable data, from which it is possible to arrive at a likely cause.

## CAUSES

- (1) *Cerebrovascular* Intracranial haemorrhage/haematoma, arterial or venous thrombosis, embolism
- (2) *Intracranial infections* Meningitis, encephalitis, cerebral malaria
- (3) *Intracranial space occupying lesion* (SOL)
- (4) *Head injury*
- (5) *Epilepsy*
- (6) *Intoxication* Poisoning with barbiturates, alcohol, organophosphorus, etc
- (7) *Metabolic* Uraemia, diabetic coma, CO<sub>2</sub> narcosis, hypoxia, alkalosis, acidosis, hyper- and hypo-natraemia
- (8) *Endocrine causes* Myxoedema, Addison's disease, pituitary apoplexy
- (9) *Late stages of cerebral degeneration*
- (10) *Physical agents* Electric shock, sunstroke, hypothermia
- (11) *Psychogenic*

## METHOD OF INQUIRY

(1) *Type of onset* Onset is *sudden* in cerebrovascular accidents, concussion and epilepsy, *gradual* in cholaemia, uraemia, diabetic coma and other toxae-mias, chronic subdural haematoma

Rapid onset, over a period of a few hours, suggests intracranial haemorrhage, embolism, or a toxic state

(2) *History of recent head injury* A careful examination of the cranium for recent head injury is essential in every case of coma, especially when a history is not available

(3) *Previous attacks* They suggest epilepsy, diabetic coma, or cerebro-vascular lesions

(4) *History of taking drugs* such as barbiturates or morphia Exposure to poisonous gases, like carbon monoxide

(5) *Pre-existing illnesses*, such as diabetes (hypoglycaemia or hypergly-caemia), cerebrovascular accident, renal disease (uraemia), cirrhosis(cholaemia), alcoholism, drug addiction, past history of psychiatric or organic brain disorder

(6) *Preceding symptoms*, such as fits, headaches or vomiting Meningitis or encephalitis may be preceded by headache and vomiting Recent symptoms of headache, personality change, or insidious hemiparesis may suggest a tumor

Complete absence of premonitory symptoms suggests a primary intra-cranial vascular lesion or an epileptiform attack A history of severe psycho-logical disturbance, e g depression, is suggestive of self-administered intoxica-tion with drugs, e g soporifics or sedatives

(7) *History of fever* may be obtained in intracranial infections

(8) *Exposure to heat or the sun* History of prolonged exposure to high temperatures or to the heat of the sun may be obtained in coma from sunstroke or heat hyperpyrexia

## CONSTIPATION

Constipation means an undue delay in the evacuation of faeces. This may be due to abnormal retention of faecal matter or delay in the discharge of excreta from the rectum. Other symptoms, such as abdominal discomfort or fullness and flatulence, may be associated with constipation.

One of the commonest of human ailments, particularly in civilized countries, constipation may be regarded as a normal "variant" rather than a disease, in view of its high incidence.

*Mechanism* Constipation is ascribable to a multiplicity of factors (1) Mechanical impediment to the forward movement of intestinal contents (as in carcinoma of colon) (2) Excessive peristaltic activity of the colon with spasticity. The excessive tone and segmental action within the colon prevents propagation of peristaltic waves (3) Deficient peristaltic activity, with atonia or dilatation of the colon (4) Deficient or absent defaecation reflex, a conditioned reflex acquired during infancy but frequently lost through faulty habits or neglect (5) Autonomic imbalance, between the sympathetic and parasympathetic nervous systems (6) Vitamin B-complex deficiency

Three main types of constipation are recognizable (1) *Colonic constipation*, where defaecation is normal but the passage through the colon, of excretory contents, is delayed, (2) *dyschezia*, where the colonic movement is normal but evacuation or defaecation is defective, and (3) *inadequate bulk of faeces*

## CAUSES

(1) *Simple constipation* (a) Dietary factors, lack of roughage, (b) abuse of laxatives, (c) diminished expulsive power due to weakness of abdominal muscles or of pelvic floor, and (d) atony or delay in the rectum due to bad training or lazy habits

(2) *Organic disease of upper alimentary tract* Pyloric stenosis, intestinal obstruction, deficient reflex activity of intestines due to organic disease of brain or spinal cord

(3) *Spasm or obstruction of colon* Pressure on colon from without, e.g., ovarian cyst. Use of drugs like ganglion blocking agents, opium, spastic colon

## METHOD OF INQUIRY

(1) *Nature and amount of habitual diet* There may be inadequate bulk for stimulation or insufficient intake of liquids may be responsible for undue dryness of the faeces

(2) *Use of laxatives or purgatives* over a long time

(3) *History of taking drugs* which have a constipating effect, such as opium derivatives or astringent antacids

(4) *Vomiting* in carcinoma of pyloric antrum or stenosing duodenal ulcer

(5) *Duration* Symptoms of recent constipation and indigestion in middle-aged or elderly patients should suggest malignancy of the colon

(6) *Pain* Cramp-like abdominal pain with anorexia and fullness after meals may suggest a diagnosis of spastic colon

## CONVULSIONS AND FITS

Convulsions may owe their origin to many and varied disease processes, including organic diseases of the central nervous system and systemic disease anywhere in the body

### TYPES OF CONVULSIVE SEIZURES

Convulsions or fits can be classified into several types according to the manifestations exhibited during the attack, they may call attention to the actual region of the brain involved in causation of the seizure

(1) *Jacksonian (local) seizures* occur almost exclusively in patients with organic lesions in the cortex, tumour, birth injury, infection or trauma. A focal seizure starts with twitching of one part of the body, usually the distal part of an extremity. It may start in the fingers, then spread to the forearm, arm, face and then the leg on the same side. If the movements spread to the opposite half of the body, consciousness is lost, and the clinical picture is that of a grand mal attack. The power of speech may be impaired or lost, if the convulsive movements involve the dominant side of the body.

(2) *Grand mal attacks* The majority of patients with convulsions suffer from grand mal epilepsy. In its classical form, it consists of premonitory symptoms or an aura, which is followed by an epileptic cry, loss of consciousness and general tonic and clonic movements.

(3) *Lennox's triad* These usually occur in childhood and consist of (a) *Myoclonic jerks* Sudden brief jerks localised or generalised can occur in conjunction with idiopathic grand mal in adults. The myoclonic jerking so common on dropping off to sleep is of no pathological significance. (b) *Akinetic seizures* sudden loss of tone in the muscles of the body. The patient may fall on the ground. (c) *Typical petit mal attacks*, with transient loss of contact with the environment.

(4) **Psychomotor attacks** (Temporal lobe epilepsy) In the majority, there is a brief loss of consciousness, rarely periods of mental cloudiness or amnesia lasting hours or days. The minor attacks of psychomotor epilepsy resemble those of petit mal, except that in the former, the duration of attacks is longer and the range of muscular movements greater. The aura often suggests temporal lobe attacks.

(5) **Reflex epilepsy** It is most commonly grand mal or focal, although petit mal can also occur in children. The attacks occur in response to some fixed and clearly recognised sensory stimulus, usually auditory or visual, or at times sudden loud noise or any strong interrupted light source such as television.

*Atypical seizures* Prolonged periods of mental cloudiness or automatic behaviour with complete amnesia are described as psychic equivalent attacks.

## CAUSES

(I) **Idiopathic.** Absence of organic factors

(II) **Symptomatic.**

### A Local causes

(1) *Neoplasms* Tumours, primary or secondary hydrocephalus

(2) *Inflammatory conditions of brain or meninges* Meningitis, encephalitis, neurosyphilis

(3) *Head injuries*

(4) *Congenital abnormalities* Cerebral diplegia, porencephaly, tuberous sclerosis

(5) *Degenerations* Diffuse sclerosis

(6) *Circulatory disturbances* Cerebral vascular accidents, hypertensive encephalopathy, Stokes-Adams syndrome

### B General causes

(1) *Toxins* Exogenous—alcohol, lead, cocaine. Endogenous—eclampsia, uraemia, cholaemia

(2) *Anoxemia* Asphyxia

(3) *Metabolic disorders* Alkalosis, hypocalcaemia

## METHOD OF INQUIRY

(1) *Ages* Infancy Birth injury, tetany, congenital, acute infections. Childhood Birth injury, trauma, infections, idiopathic epilepsy. Middle age Neoplasm, trauma. Old age Cerebrovascular conditions, neoplasm.

(2) *Head injury* The seizures may start any time after a head injury (usually from six months to two years).

(3) *Type of seizure* Fits, which occur in patients with cerebral lesions, are usually of grand mal type, less commonly psychomotor, and rarely of petit-mal type. A focal convulsive movement at the onset of a seizure, suggests the probability of a localized cerebral lesion.

(4) *Recurrent attacks* Recurrent convulsions are likely to occur in epilepsy, subdural haematoma, hypoglycaemic states, vascular dysfunctions, intracranial tumours, lead poisoning and hysteria.

(5) *Family history* Suggests idiopathic epilepsy.

(6) *Prodromata or aura* Unusual prodromata or aura, especially of local nature, indicates the presence of a local lesion in the brain. An olfactory or auditory aura points to the temporal area, and a uniform visual one to the occipital area.

(7) *Transient paresis* This may occur in focal epilepsy (Todd's paralysis).

(8) *Other neurologic symptoms* Symptoms, such as headache, localized paralysis and mental changes, should be inquired into.

(9) *Factors which precipitate seizures* such as fever, may prove important in diagnosis.

(10) *Past history of infections, illnesses and other systemic diseases*, which may be associated with cerebral lesions or sequelae.

## COUGH

The act of coughing is a defence mechanism that helps to keep the lower respiratory passages clear, protects them against the entry of foreign materials from outside, and prevents stagnation of secretions within the air passages.

Cough is a sudden and variable expiratory thrust of air from the lung and through the air passages, associated with phonation, which momentarily interrupts the physiological pattern of breathing. It may be slight, moderate or severe, occasional or paroxysmal, recurrent or continuous, transitory or persistent, painful or painless, dry (unproductive, useless) or loose (associated with expectoration). The intensity of a cough is not necessarily in keeping with the severity of the causative disease. For instance, the cough may be slight or negligible in bronchogenic carcinoma, whilst in functional disorders or hysteria, it may prove most distressing.

Whilst cough with expectoration usually suggests involvement of the lungs, bronchial tubes or upper respiratory passages, a dry unproductive cough is indicative of congestion of pharynx or larynx, early disease of lung-tissue, pleural involvement or reflex irritation. Of the five major or cardinal symptoms of respiratory disease, viz cough, expectoration, dyspnoea, haemoptysis and chest pain, cough is unquestionably the commonest.



*Mechanism* The *afferent* pathway includes the sensory end organs within the pharynx, larynx, trachea and bronchi, and sensory fibres within the ninth and tenth cranial nerves. The act of coughing, usually comprises of a sudden and severe or violent expiration after a deep inspiration and closure of the vocal cords, and may be associated in severe cases with bronchospasm or glottic spasm, resulting in dyspnoea, cyanosis, anoxia and (very rarely) death.

*Types of coughs* Many different and at times, highly characteristic types of cough, may be identified, e.g. (1) the dry and *irritable* cough of early pulmonary tuberculosis, maximal on waking up and in the early night, (2) the dry and *hawking* cough of chronic pharyngitis, laryngitis, tracheitis or neurosis, (3) the dry, *nocturnal* cough of chronic pharyngitis and enlarged uvula, (4) the dry, *reflex* cough of diaphragmatic irritation, peritonitis, gastritis, ear diseases and pericarditis, (5) the *barking* cough of hysteria or nervousness, (6) the *gander* or *brassy* or *metallic* cough of mediastinal tumours or aortic aneurysms, (7) the short and suppressed cough of pleurisy or acute lobar pneumonia with pain, (8) *paroxysmal* cough of whooping cough, bronchial spasm or carcinoma. The most distinctive of all coughs is that encountered in pertussis, because of the characteristic sound of the whoop, (9) *smoker's cough* may occur at any time since here respiratory tract inflammation is chronically present, it is characteristically provoked by smoking.

## CAUSES

(1) *Infections of respiratory tracts* (a) Acute Laryngitis, tracheitis, bronchitis. (b) Chronic Chronic laryngitis, bronchitis, bronchiectasis, pulmonary tuberculosis.

(2) *Mechanical irritation of respiratory tract* Foreign body, inhalation of irritant gases, smoker's cough, pneumoconiosis, chronic sinusitis, bronchogenic carcinoma.

(3) *Extrapulmonary (non-respiratory) causes* These induce cough through pressure on the trachea or bronchus, penetration or infiltration of the respiratory tract or through secondary involvement of lung parenchyma (e.g. pulmonary congestion or infarction). Aortic aneurysm, dilated left atrium (mitral stenosis), massive enlargement of heart, enlarged mediastinal lymphnodes, mediastinal tumours, substernal thyroid, diseases of pleura, diaphragm or oesophagus, left ventricular failure.

(4) *Reflex causes* Irritation of vagus nerve, foreign body or wax in the auditory meatus.

(5) *Psychogenic causes* Mental or emotional tension, insanity.

## METHOD OF INQUIRY

(1) *Onset* Whether sudden or insidious? The abrupt onset of a paroxysmal cough, especially in children, may be due to the presence of a foreign body in the larynx or bronchus. Severe cough may result from the inhalation of irritant gases.

(2) *Character of cough* A harsh, hoarse, or croupy cough suggests a laryngeal infection, with destructive lesions of the vocal cord, however, such as tuberculous ulceration or new growth, the cough becomes a toneless whisper. A high-pitched brassy cough (*gander* or *bovine* cough, *leopard's growl*) may result from pressure on the lower part of the trachea, malignant tumours, such as bronchial carcinoma or new growth of the thymus gland, from recurrent laryngeal nerve paralysis as in case of aortic aneurysm, large pericardial effusion, or dilated left atrium of mitral stenosis, or from a mediastinal swelling. Occasionally, a dramatic, loud and barking cough may be meant to draw attention and therefore hysterical.

(3) *Dry or productive* A persistently dry cough may be due to pulmonary tuberculosis, upper respiratory tract infection such as granular pharyngitis, post-nasal discharge associated with sinus infection, hypertrophied uvula or latent middle ear infection. Persistent expectoration is the criterion by which chronic bronchitis is defined. However it is also a common symptom in bronchiectasis, tuberculosis, bronchial asthma and chronic left heart failure especially mitral stenosis.

(4) *Sputum* Its colour and consistency should be inquired about. Truly purulent sputum indicates bacterial infection.

(5) *Does it occur in spasmodic attacks?* A paroxysmal cough may occur in whooping cough, asthma, in certain types of bronchitis and pulmonary oedema. When a paroxysmal cough is associated with unilateral wheezing, a neoplasm or endobronchial disease should be suspected. In the later stages of pulmonary tuberculosis, the cough may be productive and paroxysmal. Cardiac cough may be paroxysmal, occurring especially after exertion.

(6) *Is it painful and distressing?* A dry, short and sharp cough, obviously producing pain and suppressed to avoid the pain, indicates some lesion involving the lung parenchyma or pleura, for example pneumonia or pleurisy.

(7) *Relation to posture, time of day and meals* A productive cough aggravated by change of position always suggests pulmonary suppuration such as abscess or bronchiectasis. An abnormally long uvula may cause mechanical cough when the patient is recumbent. A cough that comes on mostly at night and is relieved by elevating the head may be due to congestive cardiac failure, lung or mediastinal tumour, bronchial allergy or diaphragmatic hernia. Sinusitis also may cause a night cough, with a tickling sensation in the back of the throat. The cough of tuberculosis, allergy or bronchiectasis is apt to be most

troublesome in the morning. A cough increased by deglutition suggests a developmental anomaly, such as a broncho-oesophageal fistula, whilst a cough soon after meals may suggest chronic lung disease.

A characteristic feature of sinus infection, associated with chronic bronchitis, is that the cough and sputum are most marked in the early morning, in mild cases, there may be little or no sputum during the day.

(8) *Associated features* Cough due to inhalation of certain types of dust is usually occupational (pneumoconiosis). Dysphagia or change of voice suggests pressure on the oesophagus, trachea or main bronchus.

## DIARRHOEA

The term diarrhoea indicates the evacuation of loose, liquid or unformed stools and is usually due to an abnormally rapid passage of food residue through the intestinal canal. As a rule, the number of stools and the bulk of faecal matter per day are more in diarrhoea.

Diarrhoea may be caused by (1) organic disease of the alimentary tract, (2) excessive mechanical or chemical stimulation of peristalsis, or (3) a hyper-sensitive neuro-muscular mechanism, controlling the intestinal movements. An incomplete evacuation of hard and scybalous masses in cases of constipation may lead to several attempts at evacuation per day (pseudo-diarrhoea), but the stools are well-formed and hard. Diarrhoea may be acute, of sudden onset and short duration, or chronic, usually of gradual or insidious onset and long duration, often extending over years. When diarrhoea is associated with the passage of blood, mucus and pus (frequently), the condition is usually spoken of as *dysentery*.

### CAUSES

#### Acute diarrhoea

- (1) *Bacterial food poisoning* due to living micro-organisms such as salmonella, *Cl. Welchii*, staph. aureus, vibrio cholera.
- (2) *Viral food poisoning* as in summer diarrhoea.
- (3) *Chemical poisoning* due to accidental ingestion of inorganic poisons such as fungi or poisonous mushrooms.
- (4) *Food allergy* due to allergy to sea foods.
- (5) So-called *travellers' diarrhoea* of obscure etiology.

#### Chronic diarrhoea

- (1) *Gastrogenous* (a) Achlorhydria or hypochlorhydria (b) *Gastric operations* Vagotomy, gastrectomy, gastrojeuno-colic fistula.

(2) *Intestinal diseases and operations* (a) Chronic intestinal infection such as amoebiasis, bacillary dysentery, actinomycosis (b) Parasitic causes like giardia lamblia or helminths e.g. strongyloides (c) Absorption defects Malabsorption syndromes (d) Operations Extensive resection or short circuit operations (e) Deficiency states Pellagra (f) Intestinal carbohydrates dyspepsia (g) Ulcerative conditions Intestinal tuberculosis, ulcerative colitis, Crohn's disease (h) Functional colonopathies Cathartic colon due to prolonged use of purgatives, mucous colitis (i) Chronic intestinal ischaemia (mesenteric artery insufficiency) (j) Carcinoid tumour (k) Diverticulitis

(3) *Pancreatic diseases and operations* Total pancreatectomy, chronic pancreatitis, Zollinger-Ellison syndrome (hyperchlorhydria, diarrhoea and fulminating peptic ulcer)

(4) *Biliary fistula*

(5) *Reflex* Pelvic inflammatory, urinary tract or appendicular disease

(6) *Constitutional disorders* Hyperthyroidism, diabetes mellitus, gastrointestinal allergy, collagen disease

(7) *Organic neurological disease* Tabes, intracranial disease

#### METHOD OF INQUIRY

(1) *Age* Early life parasitic, dysentery, abdominal tuberculosis, idiopathic steatorrhoea Middle and old age colonic carcinoma, abuse of purgatives

(2) *Sex* Colonic neurosis, hyperthyroidism and use of purgatives more common in women, colonic carcinoma in men

(3) *Constipation alternating with diarrhoea* may suggest carcinoma of colon, colonic neurosis, gastrogenous diarrhoea, laxative habit or diverticulitis

(4) *Relation to ingestion of food* Diarrhoea of gastric origin is usually in morning and immediately after each meal Diarrhoea repeated after certain foods suggests allergy

(5) *Abdominal pain* Colicky abdominal pain associated with bouts of abdominal distension and vomiting indicates small bowel obstruction e.g. Crohn's disease Dull aching or colicky abdominal pain poorly localised, constantly occurring after every meal but lacking the periodicity of ulcer pain is suggestive of chronic intestinal ischaemia

(6) *Duration* Long duration of diarrhoea may rule out malignancy as the cause

(7) *Tenesmus* or painful and recurring urge to defaecate may occur in bacillary dysentery, tuberculosis, ulcerative colitis or diverticulitis

(8) *Abdominal distension* may be complained of in malignant disease or intestinal tuberculosis

(9) *Nature of faeces* Description as given by the patient will be of help. Thus in steatorrhoea the stools are large, pale and pasty, mucus and blood suggest amoebic or bacillary dysentery, whereas large amounts of mucus will be passed in mucous colitis or ulcerative colitis. Bloody diarrhoea indicates presence of ulceration (colitis or ulcerating tumour). Frequent but normally formed stools are suggestive of functional diarrhoea. Normally formed stools preceded by passage of mucus are indicative of spurious diarrhoea in association with some degree of colonic obstruction.

Frequent, soft, non-fatty stools may suggest diarrhoea of gastric origin.

## DYSPHAGIA

Difficulty in swallowing may result from painful, mechanical or nervous interference with the mechanism of deglutition. The abnormal sensation of obstruction may be localized by the patient in the upper, middle or lower part of the oesophagus.

### CAUSES

(1) *Inflammatory lesions or mechanical defects* involving tongue, mouth, pharynx or larynx, e.g. stomatitis, cleft-palate.

(2) *Upper oesophageal dysphagia* Carcinoma (post-cricoid), pharyngocele, diphtheria, retro-pharyngeal abscess, syphilitic stenosis, myasthenia gravis, bulbar palsy, iron deficiency anaemia (Plummer-Vinson syndrome).

(3) *Mid oesophageal dysphagia* Carcinoma, extrinsic pressure from aneurysm or mediastinal tumour, foreign body, congenital stenosis.

(4) *Lower oesophageal dysphagia* Achalasia of the cardia, peptic ulcer of oesophagus, oesophagitis, simple stricture, carcinoma of oesophagus, carcinoma of fundus of stomach.

### METHOD OF INQUIRY

(1) *Age* First decade Congenital atresia or stenosis, achalasia. Second decade oesophagitis from accidental drinking of corrosive substance, congenital stenosis, achalasia. Third decade achalasia. Fourth and fifth decades syndrome of iron deficiency anaemia of females. Fifth decade onwards peptic ulceration of oesophagus, carcinoma of oesophagus, pharyngocele.

(2) *Duration* A long history, with intermittent symptoms suggests achalasia of cardia, progressive persistency indicates stenotic causes.

(3) *Precipitating factors* Factors such as the swallowing of a foreign body or an emotional upset.

(4) *Relation to solids and liquids* Equal difficulty in swallowing solids and liquids suggests an almost complete mechanical obstruction. Dysphagia, confined to solids, suggests a mechanical defect, with partial stenosis.

(5) *Time of occurrence during act of swallowing* When difficulty in deglutition arises synchronously with the passage of a bolus from the mouth into the pharynx and upper oesophagus, an abnormality of the pharynx is likely. When abnormal deglutition is associated with strangulation, coughing or nasal regurgitation, a central lesion of bulbar type, myasthenia gravis, involvement of 9th and 10th cranial nerves or a pharyngeal paralysis following diphtheria should be thought of. Lesions within or around the larynx may affect the beginning of deglutition. In lesions of the thoracic oesophagus or of extra-oesophageal tissues, the dysphagia may start two to five seconds after the act of swallowing. Lower oesophageal dysphagia usually occurs five to fifteen seconds after swallowing, in the form of discomfort in the region of ensiform cartilage.

(6) *Associated symptoms* Body wasting, sudden and severe, is in favour of new growth. Aphonia, cough and dyspnoea, when present, suggest mid-oesophageal causes. Foetor of breath and vomiting of blood may be complained of in cases of oesophageal pouch or mural new growth.

## D Y S P N O E A

Abnormal breathing characterized by increased respiratory effort and associated with distress, discomfort or a subjective sensation of "air hunger" is referred to as dyspnoea. It should be remembered that dyspnoea is not tachypnoea (rapid breathing) which refers to increased ventilation in proportion to increased metabolism, or hyperventilation when increased ventilation is in excess of metabolic needs.

### GRADES OF D Y S P N O E A

*Slight* Symptoms produced by more than average physical activity such as running, playing games.

*Moderate* Breathlessness on ordinary activity e.g. walking at average pace or up two flights of stairs or any form of manual labour.

*Considerable* Symptoms develop with less than ordinary physical activity and force patient to walk slowly on the level.

*Gross* Patient is totally incapacitated.

### C A U S E S

#### A Exertional dyspnoea

(1) *Simple hyperventilation* e.g. anaemia, pregnancy

(2) *Early heart disease* e.g. mitral stenosis

- (3) *Obstructive airways disease* Bronchial asthma, chronic bronchitis
- (4) *Restrictive lung disease* Pulmonary fibrosis, kyphoscoliosis

## B Dyspnoea at rest

(1) *Acute infective or mechanical conditions* Pneumothorax, pleural effusion, pneumonia, pulmonary infarction

- (2) *Paroxysmal dyspnoea* Acute left ventricular failure, bronchial asthma
- (3) *Metabolic causes* Acidosis of uremia or diabetes
- (4) *Psychogenic* Hyperventilation syndrome
- (5) *Cheyne-Stokes breathing* e.g., in cerebro-vascular disease

Recent or sudden dyspnoea occurring in a patient in previous good health is more commonly due to left ventricular failure or at times pulmonary embolism, spontaneous pneumothorax especially in young patients, or rarely oedema or spasm of larynx

## METHOD OF INQUIRY

(1) *Onset* Sudden onset in oedema or spasm of the larynx, spontaneous pneumothorax, pulmonary collapse and infarction

(2) *Character of breathlessness* Difficulty in fetching a deep breath in dyspnoea of psychogenic origin, also in mechanical dyspnoea due to ascites

Inspiratory dyspnoea may be due to upper respiratory tract obstruction or narrowing of bronchioles as in bronchial asthma. Expiratory dyspnoea suggests spasm

(3) *Progress* In the dyspnoea of effort syndrome, there are occasions when effort does not produce dyspnoea

(4) *Periodic or recurrent* Recurrent attacks occur in bronchial asthma

(5) *Fever* Presence of pyrexia should suggest infective conditions of the lung

(6) *History of hypertension or ischaemic heart diseases* This is in favour of dyspnoea of cardiac origin

(7) *Relation to posture* If patient becomes dyspnoeic on sitting up rather than on lying down, the possibility of a left atrial myxoma, ball valve thrombus or orthostatic hypotension must be explored

(8) *Wheeze* This is a musical sound heard by patients with airways obstruction or emphysema. It is heard mainly on expiration and is produced by air blowing through narrowed bronchi. Occasionally the patient may be aware of a localised wheeze if a large bronchus is partially obstructed by a new growth, foreign body or localised inflammation

(7) *Associated features* Cough in asthma and other pulmonary conditions like tuberculosis, bronchiectasis, etc Palpitation and fatigue in effort syndrome, oedema of legs in cardiac failure and anaemia

## EPISTAXIS

Though it is a minor symptom, epistaxis merits consideration since it may be the first pointer to serious local or general disease With the exception of spontaneous bleeding, general conditions are more commonly responsible When due to local causes other than spontaneous haemorrhage the bleeding is likely to be small in amount .

### CAUSES

#### I Local.

- 1 *Spontaneous*
- 2 *Traumatic injury* to nose or base of skull, foreign body
- 3 *Nasal disease* Rhinitis sicca, diphtheria, lupus, tumors

#### II General:

- 1 *Infective fevers* Influenza, measles, typhoid
- 2 *Liver disease* Jaundice, cirrhosis of liver
- 3 *Haemorrhagic diseases* Purpura, haemophilia, scurvy, polycythemia
- 4 *Arterial hypertension*
- 5 *Venous hypertension of congestion*, Chronic bronchitis, emphysema, pertussis
- 6 *High altitude* Mountain or air sickness

### METHOD OF INQUIRY

- 1 *Age* Foreign body in infants and young children, also diphtheria or adenoids At puberty spontaneous epistaxis may occur following severe exertion
- 2 *Recurrent epistaxis* suggestive of spontaneous primary epistaxis
- 3 *History of trauma* such as fall or blow
- 4 *Nasal discharge* accompanying epistaxis suggests nasal diphtheria
- 5 *Quantity* Profuse usually in hypertension followed by a feeling of relief after the bleeding Bleeding due to local causes usually small in amount
- 6 *Fever* Infective fevers such as typhoid, influenza, glandular fever may begin with epistaxis
- 7 *Features of haemorrhagic disease* such as family history, recurrence of bleeding, haemorrhages from other sites and presence of anaemia



## EXPECTORATION (SPUTUM)

While information about the sputum can be obtained from the patient, the naked eye inspection of the specimen is of equal if not greater importance. The respiratory tract of the normal adult produces about 100 ml of sputum per day. When there is excessive production of mucus it tends to accumulate and is coughed up as sputum. Expectoration of sputum may occur in response to physical, chemical or infective insult to the mucous membrane of the bronchi.

### METHOD OF INQUIRY

(1) *Quantity* Very large quantities of sputum are produced in bronchiectasis. If such a sputum is collected in a conical glass, it tends to separate into three layers: a mucoid layer on top, a mucopurulent layer in the middle and a purulent layer at the bottom. A large amount of pus coughed up suddenly is suggestive of rupture of a lung abscess, subphrenic abscess or empyema into a bronchus.

(2) *Appearance* The nature and consistency of the sputum should be recorded. Yellowish or purulent sputum is proof of infection somewhere in the respiratory tract. A greyish or blackish sputum signifies inhaled soot or dust, whilst jet black sputum is met with in coal miner's lung (anthracosis). Prune juice and red-currant jelly appearances of the sputum have been described in cancer of the lung but cannot be considered characteristic of the disease. A greyish-brown offensive sputum, with fragments of lung tissue, is seen in the rare case of gangrene of lung. In pneumonia, the sputum tends to be rusty or khaki, whereas in pulmonary oedema a pink frothy sputum may appear. Purulent sputum is as a rule yellow, but if it has been stagnant e.g. after a long overnight sleep, it may be greenish (due to action of verdoperoxide derived from neutrophils).

(3) *Effect of change of position* In any condition characterized by one or more large cavities, with accumulations of secretion, notably bronchiectasis and lung abscess, any sudden movement from side to side or from supine to erect position (or vice versa), may be followed by vigorous coughing with copious expectoration.

(4) *Presence of blood* The sputum may contain frank blood unmixed with sputum, or the sputum may be uniformly blood-stained, or be just streaked with blood. This is an important symptom, for it implies, with very few exceptions, an organic disease somewhere in the respiratory tract. In its lesser grades, however, it may be an incidental finding. Flecks or streaks of blood in the phlegm are not uncommonly found, intermittently, in the course of chronic bronchitis.

(5) *Smell* A foul smelling sputum or foetid expectoration is often present in cases of bronchiectasis, lung abscess or gangrene, malignant growth, and broncho-pleural fistula.

(6) *Presence of foreign bodies* Large and long fibrinous casts may be rarely expectorated in fibrinous bronchitis. Hooklets of hydatid and the small yellow sulphur granules of actinomycosis can be recognized by the naked eye, when present.

## FATIGUE

Frequently defined as “progressive decrease of capacity for physical effort” or “deficient muscular capacity” *fatigue* is a complex sensation with psychic and physical components. It is liable to be mistaken for tiredness, weakness, exhaustion or drowsiness.

*Mechanism* Although encountered at times without expenditure of muscular energy, as in cases of emotional strain, anxiety neurosis and neurasthenia, fatigue usually follows excessive, undue or unusual physical effort. Fatigue after physical exertion has been variously ascribed to (1) excessive accumulation of lactic acid in muscles and blood, (2) depleted stocks of creatine in muscle-tissues, (3) defective formation of phosphagen, (4) oxygen-debt, and (5) accumulation of myotoxic substances.

### CAUSES

- (1) *Starvation* Rigid dietary restriction or anorexia
- (2) *Prolonged inactivity* Bed-ridden state, long ailments
- (3) *Surgical causes* Operations, burns, injuries, fractures
- (4) *Infections*
- (5) *Cardiovascular causes* Rheumatic, syphilitic, hypertensive, or arteriosclerotic heart disease, vascular thrombosis
- (6) *Respiratory* Chronic asthma or bronchitis, pneumonia, methaemoglobinaemia, pulmonary congestion
- (7) *Endocrine causes* Thyrotoxicosis, uncontrolled diabetes, hyperinsulinism, Addison's disease, Simmond's disease
- (8) *Neurological* Myasthenia gravis, dystrophies of muscles
- (9) *Anaemias*
- (10) *Psychogenic* Anorexia nervosa, neurasthenia

*Tiredness*, unlike fatigue which it closely resembles, seldom occurs without physical effort. *Weakness* usually arises in association with minor loss of power of muscles, secondary to neurological causes.

*Exhaustion* is a serious and severe form of fatigue, usually secondary to major psychological or physical injuries, observed after prolonged emotional shocks, severe psychasthenia, excessively prolonged bouts of physical effort, severe traumata, lack of sleep, starvation and rigors of climate. Predisposing

factors include endocrine disorders (e.g. thyrotoxicosis, Addison's disease), diabetes, cardiac and respiratory ailments and certain types of individuals with a proclivity to "extreme fatigue" or exhaustion

## FLATULENCE

Flatulence means the presence of excess of gas in the stomach or intestines, the passage of which either by belching or per rectum causes relief of symptoms. For clinical purposes a distinction can be drawn between gastric and intestinal flatulence. The latter is often accompanied by a consciousness of gurgling (borborygmi) due to peristaltic activity. Excessive distension or bloating of the abdomen through gas is referred to as meteorism or tympanitis.

### CAUSES

#### A Gastric flatulence

Excessive swallowing of air (aerophagy), pyloric obstruction, gall-bladder dyspepsia, carcinoma of stomach, hiatus hernia

#### B Intestinal flatulence

(1) Excessive production of gas by fermentation, e.g. carbohydrate dyspepsia, chronic amoebiasis, sprue

(2) Defective absorption due to venous congestion of bowel, e.g. congestive cardiac failure, cirrhosis of liver

(3) Defective elimination—severe constipation

### METHOD OF INQUIRY

(1) History of habit of belching or neurosis

(2) Duration of symptoms—short history usually in cancer

(3) Pain may occur with cholecystitis and in hiatus hernia

(4) History of amoebic dysentery

(5) *Diet* Inclusion in diet of certain amount of roughage and greasy foods aggravates gall-bladder dyspepsia. Intolerance of carbohydrates in carbohydrate intestinal dyspepsia

## FORGETFULNESS (LOSS OF MEMORY)

Memory may be defined as the ability to retain and recall past events and experiences. The three essential features of memory are registration, retention and recall. *Defects of registration* are usually due to disordered attention

e.g. in severe psychotic states or delirium *Failure of retention* seems to be the primary factor in case of diffuse organic cerebral lesions such as atherosclerosis, head injury or Korsakoff's psychosis. Here remote past is remembered, while the more recent past is forgotten. When a person fails to remember an incident or event but can remember of it having taken place when the same is described to him, the amnesia is due to *defect of recall*. This may be seen with hysteria.

## CAUSES

- (1) Neurological disorders. Epilepsy, multiple sclerosis, GPI or meningo-vascular syphilis, intracranial tumor especially involving temporal lobe
- (2) Head injury
- (3) Hysterical or psychopathic
- (4) Nutritional. Vitamin B<sub>12</sub> deficiency, pellagra
- (5) Myxoedema and hypothyroidism
- (6) Huntington's chorea

## INQUIRY

- (1) *Onset*. Vitamin B<sub>12</sub> deficiency should be kept in mind if amnesia is of recent onset. Prolonged and gradually worsening loss of memory in cerebral atrophy of old age.
- (2) *Intermittent episodes of amnesia*. Suggest temporal lobe epilepsy.
- (3) *Progressive dementia* may occur with intracranial tumor involving frontal or temporal lobe.
- (4) *Diet and alcohol*. History of alcoholism, food fadism, malnutrition or malabsorption in vitamin B<sub>12</sub> deficiency, may be associated with loss of memory for recent events.
- (5) *Head injury* may result in cerebral damage or subdural haematoma.
- (6) *Symptoms suggestive of myxoedema*. Sluggishness, sensitivity to cold, etc.
- (7) *Presence of involuntary movements*. Huntington's chorea.

## HAEMATEMESIS (AND MELAENA)

*Haematemesis* means the vomiting of frank or gross blood from a source lying between the oesophagus and duodenal bulb. The ejected blood may be bright red or dark brown in colour. When haemoglobin is converted to haematin by the acid content of the stomach, the ejected blood is dark brown ("coffee-grounds"). *Melaena* is defined as the passage of black, tarry stools.

Retention of about eight hours in the alimentary tract is required for the blood to become black or "tarry" in appearance. Stools may continue to remain "black" for 3 to 4 days even after the bleeding has stopped.

The two symptoms of haematemesis and melaena, although frequently associated, may appear singly. The occurrence of melaena without vomiting of blood suggests a lesion below the pylorus. Both symptoms are important, being indicative, as a rule, of serious disease. Neglect or delay of treatment of such symptoms may result in shock, exsanguination and death.

#### CAUSES

- (1) *Oesophageal* Varices, peptic oesophagitis, carcinoma
- (2) *Stomach* Chronic gastric ulcer, erosive gastritis as from drugs like aspirin, carcinoma, hiatus hernia, Mallory Weiss syndrome, benign tumors, foreign body
- (3) *Duodenum* Ulcer, diverticulum
- (4) *Miscellaneous* Blood dyscrasias, chronic renal insufficiency, pseudo-xanthoma and Ehlers-Danlos syndrome, polyarteritis nodosa

#### METHOD OF INQUIRY

- (1) *Age* Duodenal ulcer in young men, malignancy in older age group
- (2) *Previous episodes* of haematemesis or melaena point towards the diagnostic probability of peptic ulcer
- (3) *Intake of drugs* Haematemesis after intake of corticosteroids, butazolidin, salicylates, suggests peptic ulcer or gastritis as the most likely cause
- (4) *Addiction to alcohol* This should arouse suspicion of cirrhosis or gastritis
- (5) *Pain* History of chronic intermittent distress or pain in the epigastrium favours peptic ulcer
- (6) *Anorexia or dysphagia* should arouse suspicion of cancer of the stomach or oesophagus, especially when these symptoms are associated with rapid weight loss
- (7) *History of hypertension or bleeding disorder*

#### HAEMATURIA

Loss of blood in the urine is referred to as haematuria. Haematuria may be due to bleeding anywhere in the urinary tract from the glomerulus to the urethral meatus. It is essential to establish that the urine contains blood and not blood pigment (haemoglobinuria) or some colouring matter, such as

beet-root or rhubarb Blood may appear in sufficient quantity to produce a frankly bright red or bloody or portwine coloured urine Small quantities may produce a smoky urine or be only apparent on microscopic examination

## CAUSES

### I Local lesions.

- (1) *Kidney* Acute glomerulonephritis, pyelonephritis, papillary necrosis, infarct, tuberculosis, calculus, tumor, trauma, polycystic kidney
- (2) *Ureter* Calculus, tumor
- (3) *Bladder* Tumor, cystitis (including tubercle), calculus, trauma, foreign body, bilharziasis
- (4) *Prostate* Benign prostatic enlargement, malignancy, prostatitis
- (5) *Urethra* Trauma, tumour, calculus, foreign body

### II General disorders

- (1) *Bleeding disorders* Purpura, haemophilia, leukaemia
- (2) *Collagen diseases* Polyarteritis nodosa, lupus erythematosus
- (3) *Anticoagulant drugs*
- (4) *Subacute infective endocarditis*

## METHOD OF INQUIRY

(1) *Age* Newborn Prothrombin deficiency Child Scurvy, acute nephritis, acute leukaemia, acute infectious fevers, bladder stone, meatal ulcer  
Young adults Renal calculus, tuberculosis of kidneys, malignant tumours, hypertension, prostatic disease

(2) *Residence* Bilharziasis is common in Egypt

(3) *Administration of anticoagulant drugs*

(4) *History of trauma*

(5) *Quantity of blood* Profuse in kidney or bladder tumours or rarely in prostatic neoplasm, trauma

(6) *Relation of haematuria to act of micturition* Presence of bright red blood is consistent with haemorrhage in bladder or lower urinary tract If urine is blood stained only at the beginning of micturition, it probably means that the blood comes from the urethra or prostate Bleeding from kidneys or upper part of ureter usually results in the entire specimen being evenly stained with dark red blood If the urine passed when first emptying the bladder is clear and blood staining occurs during the final efforts to void the bladder, the haemorrhage is likely to be from the base of the bladder particularly in case of cystitis

(7) *Presence or absence of pain* Calculus in any part of the urinary tract usually gives rise to renal colic. Acute urethritis and urethral caruncle will cause pain during micturition. Carcinoma of prostate gives rise to pain in perineal region but this is not associated with micturition. Malignant growths of bladder cause pain on micturition referred to the penis and suprapubic area. Sometimes passing of a blood clot down the renal tract will give rise to severe pain and excessive numbers of oxalate crystals in the urine may cause smarting micturition and haematuria. Haematuria is painless when it occurs in enlarged prostate, renal neoplasms, congenital cystic kidneys and tuberculosis, also in systemic conditions such as purpura and hypertension.

Increased frequency of micturition will be complained of in case of a local cause in bladder, tuberculosis or pyelitis.

(8) *Associated symptoms* Puffiness of face in acute nephritis, pyrexia in pyelitis, haemorrhages elsewhere in the body in blood disorders.

## HAEMOPTYSIS

Haemoptysis signifies the expectoration of blood and is indicative of serious disease of the respiratory tract. It is necessary, however, to make sure that the blood has been coughed up and does not come from some other source, such as the nose, mouth or stomach. Blood arising from the respiratory tract is bright red and frothy, whilst the sputum continues to be blood-tinged for several days after the initial haemoptysis. Haematemesis may at times be confused with haemoptysis, the patient being uncertain of whether he has coughed up or vomited the blood. Blood from the stomach is usually clotted, darker in colour, non-frothy, contains food particles and is acid in reaction.

### CAUSES

#### A. Local causes within the respiratory tract

- (1) *Infections of lungs and bronchi* Pneumonia, pulmonary tuberculosis, bronchiectasis, *Aspergillus fumigatus* infection
- (2) *Neoplasms* Bronchogenic carcinoma
- (3) *Circulatory causes* Mitral stenosis, pulmonary infarction, aortic aneurysm
- (4) *Trauma to chest*

#### B General causes

- (1) *Blood diseases* Leukaemias, purpuras, haemophilia
- (2) *Hypertension* Systemic or pulmonary hypertension
- (3) *Haemorrhagic fevers*
- (4) *Of unknown etiology*

## METHOD OF INQUIRY

(1) *Age* Childhood likely causes are bronchiectasis, mitral stenosis or pulmonary tuberculosis Adult tuberculosis, bronchiectasis, mitral stenosis Middle and old age bronchogenic carcinoma

(2) *Quantity of blood* Profuse haemoptysis occurs most commonly in advanced cases of pulmonary tuberculosis, mitral stenosis, bronchiectasis and bronchogenic carcinoma Mild haemoptysis, with at times simply a few streaks of blood in the sputum, may occur in early pulmonary tuberculosis or acute or chronic bronchitis

(3) *Symptoms* Previous cough, fever, sputum, night sweats and loss of weight suggest tuberculosis Repeated haemoptysis, without significant deterioration of health, is in favour of bronchiectasis In pulmonary infarction, there may be a history of recent operation or of phlebitis, and the attack frequently coincides with acute pain in the chest Dyspnoea and other cardiac symptoms suggest mitral stenosis

(4) *History of trauma* such as gun-shot wounds or fracture of ribs

(5) *Family history* Haemoptysis occurring in a patient, one or more of whose relatives are known to be tuberculous, should immediately suggest this cause

(6) *Associated features* The co-existence of haemorrhages from other sites suggests a blood disease A foetid sputum is common in lung abscess and bronchiectasis An abrupt onset with fever and toxæmia suggests a specific fever

## HALITOSIS

Bad breath, halitosis or "foetor exore" is a fairly common symptom of disagreeable nature encountered in a variety of ailments of the nose, throat, lungs, stomach and mind

## MAIN CAUSES

(1) Nasal and oral Chronic nasal and nasopharyngeal catarrh or discharge, oral sepsis, dental caries or sepsis and tonsillar crypt infections

(2) Lung diseases Bronchiectasis, lung abscess, lung gangrene, pleuro-bronchial fistula

(3) Fevers and toxic ailments

(4) Gastrointestinal Chronic constipation, intestinal obstruction, colonic stasis

(5) Metabolic Acidosis, alkalosis, uræmia, high fat diet.

(6) Neuroses and psycho-neuroses



## HEADACHE

Headache or *cephalgia* is one of the commonest of all symptoms. Although of minor significance in a great many cases, it may at times be a symptom or indicator of serious organic disease.

### CAUSES

- (1) **Psychogenic headache** in neurotic and psychotic patients
- (2) **Tension headache**
- (3) **Migraine and its variants**
- (4) **Organic causes**
  - (a) *Causes in extracranial structures* (referred pain)
    - (i) *Eyes* Eye strain, glaucoma, iritis
    - (ii) *Teeth* Dental root abscess, unerupted molar
    - (iv) *Arteries* Temporal arteritis
    - (v) *Bones and joints* Cervical spondylosis, temporo-mandibular joint syndrome, Paget's disease
  - (b) *Causes in intracranial structures*
    - (i) *Space occupying lesions* Tumor, abscess
    - (ii) *Meningeal irritation* Meningitis
    - (iii) *Vascular* Occlusive cerebrovascular disease, subdural haematoma
    - (iv) *Trauma* Head injury
    - (v) *Increased intracranial pressure* Benign intracranial hypertension
    - (vi) *Lowered intracranial pressure* Post-lumbar puncture headache

### METHOD OF INQUIRY

(1) *Total duration* Headache of many years' duration, with little or no progression, is usually benign, recurring headaches from childhood are probably migrainous. A short history of intense headache suggests new growth or meningitis.

(2) *Nature and severity* Severe headache may be due to cranial, intracranial or meningeal disease. Bursting or throbbing pain occurs in intracranial tumour, abscess or hypertension. Pain of a boring type is common in hysteria and at times migraine. Paroxysms of shooting or darting pain suggest neuralgia or neuritis.

(3) *Situation or site* Unilateral headaches suggest migraine, middle ear disease, cerebral tumour or abscess, temporal arteritis or trigeminal neuralgia.

Frontal headaches suggest uraemia or eye-strain, occipital headaches suggest posterior fossa tumour, cervical osteoarthritis or sub-arachnoid haemorrhage. Vertical headaches may be psychogenic.

(4) *Continuous or intermittent* Migrainous headaches are characteristically intermittent or periodic with freedom from headaches in between attacks. A history of increasingly severe headaches suggests the possibility of an expanding intracranial lesion, such as brain tumour, subdural haematoma or aneurysm. Constant band-like pressure pain is usually psychogenic.

(5) *Time of occurrence* Early morning headache suggests intracranial tumour, migraine, hypertension, nasal sinusitis or severe anaemia. Evening headache suggests eye-strain, fatigue or mental exhaustion. Nocturnal headache may occur in cerebral tumour, abscess, meningitis or secondary syphilis.

(6) *Aggravating factors* Anxiety headache may be intensified by mental effort or a stuffy atmosphere. Headache caused by an intracranial space-occupying lesion is increased by recumbency, stooping or straining.

(7) *Family history* Similar headaches in other members of the family are common in migraine and hypertension.

(8) *Associated symptoms* Nausea, vomiting and visual disturbances in migraine. Anxiety or depression in psychogenic headache. Symptoms of nasal obstruction or discharge in sinusitis. Headache, vomiting (without nausea) and fits suggest intracranial lesion.

## HEART-BURN (OR PYROSIS)

A subjective sensation of heat, warmth or burning behind the sternum or high up in the epigastrium, usually referred to as *pyrosis*, *heart-burn* or *water-brash*, is an exceedingly common, annoying and at times refractory symptom of functional gastrointestinal disorders.

*Mechanism* Once attributed to the regurgitation of the acid contents of the stomach into the oesophagus (unlikely, since injection of acid into the gullet does not reproduce this sensation), pyrosis is nowadays regarded as a neuromuscular disorder with tonic spasm at the lower end of the oesophagus and associated or not with regurgitation of gastric contents through reversed peristalsis. It is also mentioned as a "pain-equivalent" of painful lesions within the pyloro-duodenal region.

### CAUSES

(1) *Organic lesions* Oesophagitis or ulceration, peptic ulcer, pyloric spasm, hiatus hernia.

(2) *Functional causes* Faulty dietetic habits (excess of fluids, air-swallowing, bolting of food), alcohol, smoking, chewing gum.

(3) *Psychogenic Neurosis*, repressed emotions (so called "touchy" or hypersensitive individuals)

## HICCUP

A recurring, involuntary spasm or clonic contraction of the diaphragm, associated with a characteristic sound (due to violent sucking in of air through a narrowed laryngeal orifice because of approximated vocal cords) is described as a *hiccup*, *hiccough* or *singultus*. Whilst *afferent* impulses from various structures of the body are conveyed to the respiratory centre by the vagus and phrenic nerves, *efferent* stimuli travel down to the diaphragm via the phrenic nerves.

### CAUSES

These are many and varied. They may be gastrointestinal, toxic, neurological, psychogenic, infective or surgical.

(1) *Intraabdominal causes* Peritonitis, diaphragmatic lesions, intestinal obstruction, subphrenic abscess, dilatation of stomach or liver abscess.

(2) *Mediastinal causes* Enlarged heart, pericardial effusion, mediastinal tumours, aortic aneurysm, mediastinitis, asthma, substernal goitre.

(3) *Toxic causes* High fevers, toxæmia, septicaemia, shock.

(4) *Neurological* (through irritation of respiratory centre) Tuberculous meningitis, encephalitis, hydrocephalus, epilepsy, chorea, cerebral arteriosclerosis, brain tumour.

(5) *Psychogenic Neurosis*, hysteria, sudden laughter, swallowing cold drinks, hot drinks, cold shower.

(6) *Post-operative* Dilatation of stomach, peritonitis.

(7) *Metabolic and avitaminosis* Uraemia, diabetic acidosis, gout.

(8) *Epidemic hiccup* Viral infection in males over 40, usually in epidemics related to influenza and encephalitis and followed (at times) by Parkinsonian manifestations.

## HOARSENESS OF VOICE

A voice that is rough, harsh and lower in pitch than normal is usually described as hoarse. The change in quality of the voice may range from a slight roughness of tone or huskiness to hoarseness or complete aphonia. Hoarseness is usually indicative of some interference with the phonatory function of the larynx.

## CAUSES

- (1) *Inflammatory lesions* Acute laryngitis, subacute or chronic laryngitis laryngeal diphtheria, tuberculosis of larynx, syphilis of larynx
- (2) *New growths* Benign papilloma or fibroma, haemangioma or malignant disease
- (3) *Laryngeal paralysis* Paralysis of recurrent laryngeal nerve, bulbar lesions, pressure of cervical or intrathoracic lesions

## METHOD OF INQUIRY

- (1) *Onset* Acute febrile onset suggests infectious laryngitis or diphtheria
- (2) *Duration* Hoarseness associated with acute respiratory infection is self-limited and does not last for more than two to three weeks
- (3) *Occupation* A vocal or singer's nodule, in one who uses the voice excessively, may cause huskiness of voice
- (4) *Relation to use of voice* A characteristic feature of chronic laryngitis is hoarseness and weakness of the voice most marked in the early hours of the day, with improvement as the day progresses
- (5) *Pain* Tuberculosis of larynx frequently causes pain localized to the laryngeal area or referred to adjacent structures, such as the ear
- (6) *Surgical or accidental* Hoarseness after operation or trauma suggests injury to the recurrent laryngeal nerve
- (7) *Dyspnoea* The coexistence of dyspnoea suggests bilateral pulmonary tuberculosis, mitral disease, aortic aneurysm or pericardial effusion
- (8) *Aphonia* When associated with a complete loss of voice, hysterical aphonia should be suspected, especially if the patient is able to produce a normal sound on coughing

## IMPOTENCE

Impotence is an inability to achieve penile erection

## CAUSES

- (1) *Of psychogenic origin* in majority
- (2) *Endocrine disorders* (a) Diabetes mellitus is probably the commonest organic disease related to impotence (b) Others Simmond's disease, acromegaly, Cushing's syndrome, hypo or hyper-thyroidism Addison's disease or associated with hypogonadism
- (3) *Cirrhosis of liver*

(4) *Neurologic diseases* Tabes, multiple sclerosis, cauda equina or conus medullaris lesions, subacute combined degeneration, dystrophia myotonica  
Temporal lobe tumor

(5) *Drugs* Phenobarbitone if taken for long, bromides, morphine, cocaine, alcohol, guanethidine

## INTERMITTENT CLAUDICATION

Intermittent claudication (claudication = limping) implies a clinical symptom of pain in the lower limbs brought on by walking, and relieved by rest. The symptom is more common in males and increases in incidence after middle age.

### CAUSES

(1) *Arteriosclerotic vascular disease*

(2) *Bone or joint disease* Paget's disease of bone, osteomalacia, osteoarthritis, malignant deposits, spondylolisthesis, midline protrusion of lumbar disc

(3) *Neurogenic* due to lesions of cauda equina, arachnoiditis, hypertrophic interstitial neuritis, congenital stenosis of lumbar neural canal

### INQUIRY

(1) *Duration of discomfort or pain* In claudication due to bone or joint disease patient may not have to stop and may be able to 'walk off the pain'. If pain increases with exercise so that the patient has to stop and passes off usually in ten minutes it is either vascular or neurogenic.

(2) *Pain only on standing* This is unlikely to be vascular and the lesion is usually spondylolisthesis or one of the bone and joint disorders.

(3) *Relief of pain* On stretching or twisting of the back after walking suggests spondylolisthesis.

(4) *Distribution of pains* Commonly vascular pain is felt in the middle of the calf and neurogenic pain on its lateral side. Pain restricted to lateral edge of foot is likely to be neurogenic, but pain across the whole foot is usually vascular and is particularly characteristic of Buerger's disease, occlusion or other causes of small artery disease.

(5) *Accompanying symptoms* If paraesthesiae as well as pain occur on walking the lesion is most likely to be in cauda equina.

## JAUNDICE

Jaundice or icterus is a condition characterized by yellowish discoloration of the tissues by bile pigment. Since bile pigment has an affinity for elastic tissue, the skin, ocular sclera and blood vessels, which contain many elastic fibres, become particularly yellow.

## CAUSES

Three main varieties of jaundice are

## A Hepatocellular jaundice (Toxic-infective or necrotic jaundice)

- (1) *Acute* Virus hepatitis, Weil's disease
- (2) *Chronic* Cirrhosis of liver

## B Obstructive jaundice

- (1) *With extrahepatic obstruction* Carcinoma of ampulla, gall-stone obstruction, fibrous stenosis of duct.
- (2) *Without extrahepatic obstruction*
  - (a) *Acute* Drugs like PAS, chlorpromazine, organic arsenicals, methyl testosterone, butazolidin
  - (b) *Chronic* Primary biliary cirrhosis, malignant deposits in liver

## C Haemolytic jaundice

- (1) *Congenital* Spherocytosis
- (2) *Acquired* Incompatible blood transfusion

Since jaundice is due to increase of bilirubin in the blood, it may arise in one of four ways

(a) *Increased bilirubin load* on the liver cell Haemolytic jaundice In haemolytic states, haemoglobin is released from the red blood cells in excessive quantities, causing a rise in serum bilirubin values

(b) *Disturbed bilirubin transport* Familial non-haemolytic jaundice (Gilbert's disease) Bilirubin diffuses into the cells from the sinusoids and is actively transported to the microsomes for conjugation

(c) *Disturbed conjugation of bilirubin* Neonatal jaundice, congenital familial non-haemolytic jaundice with kernicterus (Crigler-Najjar type) In neonatal jaundice, there is deficiency of bilirubin conjugating enzymes

(d) *Disturbed bilirubin excretion*

(i) *Intrahepatic cholestasis* Chlorpromazine jaundice Defect lies between the microsomes and the main bile ducts

(ii) *Extrahepatic cholestasis* Carcinoma of pancreas Defect lies within the major bile ducts

## METHOD OF INQUIRY

## I History

(1) *Age and sex* Infective hepatitis is common in young adults, common duct stone and neoplastic jaundice in middle-aged or elderly individuals

Portal cirrhosis, primary cancer of liver and pancreatic cancer predominate in the male, while common duct stone, primary biliary cirrhosis and carcinoma of gall-bladder are commoner in the female

(2) *Occupation* Any employment involving handling of hepatotoxic agents like DDT, heavy metals, beryllium, etc should be inquired. There is a predisposition to Weil's disease among workers in rat-infested premises

(3) *Contact* with jaundiced patients, if recent, should suggest possibility of infectious hepatitis

(4) *Family history* Association with anaemia, gall stones or removal of spleen may suggest haemolytic jaundice

(5) (a) *Past history of recent biliary tract surgery* A recent gall-bladder operation leads to suspicion of residual common duct stone or stricture

(b) *History of alcohol intake* in cirrhosis

(c) *Use of drugs* such as chlorpromazine or methyl testosterone may cause intrahepatic cholestasis

## II Symptoms

(1) *Onset of jaundice* Sudden suggests virus hepatitis or gallstones; gradual more likely with cirrhosis, pancreatic carcinoma or metastases; progressive typical of malignant obstruction. Fluctuating with stone in common bile duct, carcinoma of ampulla of Vater or repeated haemolytic episode

(2) *Abdominal pain* Present painful jaundice strongly suggests gallstones or pancreatic disease. Severe boring pain passing through the back suggests pancreatitis or pancreatic tumor. Absent in virus hepatitis. In older patients painless but fluctuating jaundice suggests intermittent obstructive gall stones or a necrosing papillary carcinoma. Painless but progressive jaundice is usually due to malignant obstruction of common bile duct

(3) *Fever and chills* likely to be due to cholangitis

(4) *Pruritus* characteristic of cholestasis

(5) *Weight loss* more likely in malignancy

(6) *Urine* Dark urine indicates cholestatic or hepato-cellular jaundice

(7) *Stools* Pale stools indicate cholestatic jaundice

## NAUSEA

By nausea is meant a peculiar psychic disturbance associated with a "feeling of impending vomiting" or "an imminent desire to vomit." Frequently a precursor of vomiting, nausea is a complex sensation felt in the throat, epigastrium, and associated with vasomotor or autonomic manifestations.

(e.g. bradycardia, hypotension, sweating, lassitude, headache and dizziness) It may be triggered off by an unpleasant odour or sight (e.g. faecal smell or sight of blood)

The *mechanism* of nausea, although accredited at times to abnormal intestinal motility, reverse peristalsis of the duodenum and gastric hypomotility or hyposecretion, continues to remain obscure. Nausea can be induced by apomorphine even after denervation of the intestine or evisceration.

Nausea *without vomiting* may occur in jaundice, chronic gastritis, carcinoma of stomach, irradiation treatment, severe anaemia or renal insufficiency. Its association with *vertigo* suggests labyrinthine disease, and with *headache*, migraine. *Vomiting without preceding nausea* can occur in pyloric stenosis, projectile vomiting of increased intracranial pressure and in hysterical vomiting. *Morning nausea* is common during pregnancy, toxæmias, and in gastritis and alcoholism.

## OEDEMA

By the term oedema is meant an abnormal or excessive accumulation of fluid in the tissue spaces of the body. Oedema may be *localized* or *generalized* and either confined to the subcutaneous tissues of the body or associated with transudates into serous sacs (pleural, peritoneal or pericardial).

Oedema is due to general or local transfer of fluid from the blood plasma of capillaries into the tissue spaces in abnormal amounts. Subcutaneous oedema can be usually recognized by digital pressure, which leads to "pitting" of the skin and subcutaneous tissues.

### CAUSES

#### A Generalized oedema (Anasarca, dropsy)

- (1) *Cardiac failure*
- (2) *Renal diseases* Nephrosis, acute nephritis
- (3) *Hypoproteinaemia*
- (4) *Anaemia*
- (5) *Vitamin deficiency* Beriberi
- (6) *Malnutrition* Starvation, anorexia nervosa, war-oedema, famine oedema
- (7) *Cachexia* Malignancy, advanced tuberculosis
- (8) *Epidemic dropsy*
- (9) *Hepatic oedema*
- (10) *Myxoedema*



**B Localized oedema**

- (1) *Inflammatory* Cellulitis, abscess, insect bites
- (2) *Traumatic* Fracture, sprain
- (3) *Allergic* Angioneurotic oedema
- (4) *Venous obstruction* Thrombosis or pressure on vena cava, varicose veins
- (5) *Congenital* Milroy's disease (hereditary trophoedema)
- (6) *Lymphoedema* Filariasis, post-operative
- (7) *Irritants* Mechanical, chemical or thermal

**METHOD OF INQUIRY**

(1) *Age and sex* Renal oedema more common in children Congenital oedema occurs almost exclusively in females Higher incidence of thrombosis of varicose veins in females

(2) *Family history* Oedema occurring in several members of the family or in several individuals of same locality in epidemic dropsy or nutritional oedema Familial tendency in Milroy's disease

(3) *Onset* Sudden in acute nephritis

(4) *Site and distribution of oedema* Renal oedema starts as puffiness of face, cardiac and hepatic oedema in dependent parts of the body In cirrhosis, oedema follows distension of abdomen from ascites, in other conditions oedema precedes the ascites

(5) *Dietary history* Starvation and malnutrition in hypoproteinaemia and beriberi

(6) *History of bleeding* Bleeding piles suggest anaemia or cirrhosis, ankylostomiasis suggests anaemia

(7) *Symmetrical or asymmetrical* Localized oedema, either inflammatory or due to venous or lymphatic blockage, tends to be asymmetrical and is usually confined to one extremity

(8) *Fever* Recurrent attacks of fever with rigors suggest filariasis

(9) *Associated features* Oliguria and smoky urine in acute nephritis Orthopnoea and perhaps cyanosis in cardiac oedema Pallor and dyspnoea in anaemia Other evidences of B-complex deficiency such as sore tongue and pigmentation in beriberi Urticaria or other manifestations of allergy in angioneurotic oedema Gastrointestinal symptoms in epidemic dropsy Anorexia and perhaps haematemesis in cirrhosis of liver Chest pain, cough and dyspnoea in mediastinal obstruction due to tumour

## PALPITATION

By palpitation is meant consciousness of the heart beating. It has been defined as a "conscious appreciation of the heart's action". Although popularly regarded as a sign of heart disease, palpitation is more often than not associated with conditions outside the heart.

The heart's action in palpitation may be unduly fast or slow, regular or irregular, forcible, weak or normal.

### CAUSES

#### A Extrinsic or extracardiac causes

- (1) Psychosomatic factors Emotional excitement, fear or injury
- (2) During convalescence from fevers, malaria, typhoid, influenza or diphtheria
- (3) Neurocirculatory asthenia in conjunction with exhaustion, sighing, inframammary pain, sweating and faintness
- (4) Physiological, during exertion, excitement or fever
- (5) Addictions, drugs and poisons (toxic) Tea, coffee, tobacco, alcohol, ephedrine, digitalis, thyroid, atropine, adrenaline, coal gas, car fumes, etc
- (6) Abdominal causes Gastric flatulence, chronic amoebiasis, diaphragmatic hernia, ascites, tympanitis, pregnancy
- (7) Intrathoracic causes Lung collapse or fibrosis, pneumothorax, pleural effusion, aortic aneurysm, mediastinal tumour
- (8) Thyrotoxicosis
- (9) Disorders of the blood Anaemias and polycythaemia

#### B Intrinsic or intracardiac causes

- (1) Ectopic rhythm Extrasystoles, paroxysmal tachycardia, atrial fibrillation or flutter
- (2) Stokes-Adams syndrome
- (3) Hypertension
- (4) Mitral or aortic valve disease
- (5) Congenital heart diseases

### METHOD OF INQUIRY

(1) *Onset* Onset is insidious in valvular disease like aortic regurgitation or mitral stenosis. Sudden onset in paroxysmal tachycardia.

(2) *History of rheumatic fever* suggests valvular disease of heart.

(3) *History of taking drugs* like adrenaline, ephedrine, thyroid extract or atropine Excessive consumption of tobacco, coffee or tea may be responsible for a cardiac arrhythmia like extrasystoles

(4) *Description of palpitation* Sensation of heart stopping for a beat followed by a sudden throb in the neck or throat is in favour of extrasystoles

(5) *Periodicity* Recurrent attacks, with absence of symptoms in between attacks, common in paroxysmal tachycardias

(6) *Fever* Pyrexia itself may cause palpitation due to tachycardia, especially in sensitive individuals

(7) *Duration* Paroxysmal tachycardia and extrasystoles may last for seconds or continue for hours or days

(8) *Relieving factors* Certain postures or manoeuvres adopted by the patient in the past, such as holding of the breath or lowering of the head to stop attacks of palpitation, are highly suggestive of paroxysmal atrial tachycardia.

(9) *Associated features* Fatigue, sighing respiration and inframammary pain in effort syndrome Tremors, loss of weight and emotional instability in thyrotoxicosis, dyspnoea in cardiac failure cough and expectoration in lung and pleural diseases

## PRURITUS

Pruritus or itching can be defined simply as a desire to scratch It is the single most important dermatological symptom

### CAUSES

*Generalised pruritus* Dry skin, allergic, psychogenic, pregnancy, diabetes mellitus, uraemia, lymphomas particularly Hodgkin's disease, internal malignancies, carcinoid syndrome, tissue anoxia

*Local pruritus* *Pruritus ani* (a) Mechanical causes haemorrhoids, fissure, fistulas, diarrhoea. (b) Infections Thread worms, yeast (*candida albicans*), fungi (c) Other causes Vitamin A or B complex deficiency, psychogenic, associated with generalised pruritus After tetracycline therapy

*Pruritus vulvae* (a) Mechanical causes Secondary to vaginal discharge, congestion (b) Metabolic oestrogen deficiency (causing a dry, atrophic skin), diabetes mellitus, vitamin A deficiency (c) Infections *candida*, secondary to vaginitis

## REGURGITATION OF FOOD

This is a common but neglected symptom of gastrointestinal disorder or disease Regurgitation of bile-stained, bitter liquid into the mouth, associated with burning pain behind the sternum (*heart-burn*), is referred to as pyrosis or "water brash"

## CAUSES

(1) *Neurological causes* Paralysis of soft palate (e.g. postdiphtheritic paralysis), bulbar palsy, encephalitis, myasthenia gravis, hysteria. This results in regurgitation of drink or food through the nose.

(2) *Oesophageal causes* Obstruction, carcinoma, spasm, pressure from without.

(3) *Gastric causes* Dyspepsia, hyperacidity, ulceration, carcinoma, pyloric obstruction.

(4) *Psychogenic* Excessive laughter or vomiting, hysteria, neurosis.

(5) *Miscellaneous* e.g. cleft-palate.

## METHOD OF INQUIRY

(1) History of emotional disorder, neurosis, diphtheria, neurological features.

(2) Loss of appetite and weight.

## SNEEZING

A fairly deep inspiration followed by a massive and uncontrollable expiration of air through the nose, associated with a characteristic sound, is usually referred to as a *sneeze*. Sneezing is a useful compensatory mechanism employed by the body in its attempt to get rid of noxious, toxic or irritating particles, substances or gases from within the nose. In excess, however, sneezing can prove highly annoying, exhausting or even incapacitating to the patient. In the reflex act of "sneezing", sensory impulses are transmitted from the olfactory end-organs of the nose, through the sensory fibres of the trigeminal nerves.

## CAUSES

Sneezing may be due to local causes, general or systemic diseases or psychogenic factors.

(1) *Local causes* Acute coryza or rhinitis (usually viral in origin), hay-fever, nasal polyp, deviated septum, foreign body impaction, dusts or irritating substances or gases.

(2) *General causes* Measles, influenza, whooping cough.

(3) *Drugs* Salicylates, opium, iodine or iodides.

(4) *Psychogenic* Neurosis, hysteria, psychoneurosis.

## STRIDOR

By this is meant noisy breathing with a harsh crowing sound mainly during inspiration and resulting from partial obstruction of the larynx or trachea. Stridor is usually accompanied by dyspnoea.

Also, referred to as "laryngeal stridor", stridor is a serious condition, capable of terminating fatally unless taken in hand early.

*Wheezing*, usually indicative of bronchospasm as in asthma or acute bronchitis, is much commoner and less dangerous than stridor. It is distinguishable from the latter by a different quality and pitch of the note or sound.

## CAUSES

- (1) Foreign body (impacted)
- (2) Congenital abnormalities Congenital laryngeal stridor
- (3) Inflammatory lesions Laryngitis, laryngo-tracheobronchitis
- (4) Neoplasms Papilloma, pressure from goitre, retropharyngeal abscess
- (5) Traumatic lesions
- (6) Functional disturbances of larynx

## METHOD OF INQUIRY

(1) *Duration* A short duration of the symptom suggests obstruction by a foreign body, functional disturbance of the larynx or inflammatory disease.

(2) *Mode of onset* Instantaneous onset of dyspnoea and stridor suggests foreign body impaction or neuromuscular disturbance of larynx. Acute onset is typical of laryngitis, laryngismus stridulus, measles, whooping cough or diphtheria. A slow and gradual onset suggests papilloma or paralysis of recurrent laryngeal nerve.

(3) *Age* Congenital lesion likely in an infant, a foreign body or inflammatory reaction in childhood, neoplastic disease in later years.

(4) *Pain* Pain is usually complained of when there is acute inflammation with abscess formation or the surface is broken by ulceration or injury.

(5) *Quality of voice* As a rule, a clear (even though weak) voice suggests that the lesion is below the level of the vocal cords, in the trachea. A harsh, rough and hoarse voice suggests a lesion involving the cords.

(6) *Difficulty in swallowing* Dysphagia may be encountered in retropharyngeal abscess. Difficulty in swallowing, particularly when associated with spilling over of saliva into the tracheobronchial tree, suggests a lesion of the upper oesophagus with infiltration or penetration of the tracheobronchial wall and resultant obstruction of the trachea. Coexistence of fever should suggest an inflammatory lesion. A clear history of lodging of a foreign body in the throat may be obtained.

## SYNCOPE

A sudden and transient loss of consciousness, secondary to acute decrease in blood supply to the brain, is called syncope ("fainting" or "swooning"). Syncope may be a transient or relatively benign disorder, or may reflect a progressive and life-threatening illness

### CAUSES AND MECHANISMS

#### A *Syncope due to decrease in cerebral perfusion pressure*

- (1) Vasovagal syncope (common faint)
- (2) Carotid sinus syndrome from hypersensitivity of carotid sinus
- (3) Postural syncope
- (4) Cerebral syncope from traumatic brain damage, or localised cerebral vascular disease
- (5) Cough syncope
- (6) Micturition syncope

#### B *Syncope due to inadequate cardiac output (cardiac syncope)*

- (1) Cardiac disease Aortic stenosis, pulmonary stenosis, pulmonary hypertension, ballvalve obstruction of mitral valve
- (2) Cardiac arrhythmias Attacks of paroxysmal tachycardia particularly in the elderly, high grade partial or complete heart block, a trial flutter or fibrillation
- (3) Reflex syncope Cardiac standstill occurring from reflex vagal activity e.g. fainting associated with irritation of pleura or peritoneum, or oesophagoscopy or bronchoscopy

#### C *Syncope due to metabolic causes*

Hypoxia, hypoglycaemia, hyperventilation syndrome

#### D *Hysterical syncope*

### METHOD OF INQUIRY

(1) *Age and Sex* A simple faint can occur at any age. Carotid sinus syncope is more common in middle and old age. Hysterical fainting and hyperventilation are more common in adolescent girls and young women.

(2) *Exciting causes* Postural syncope may occur after prolonged standing in fixed position or after strenuous exercise. Exertion may cause fainting from anoxic syncope, as in congenital heart disease, aortic stenosis and chronic cor pulmonale. Post-tussive syncope occurs after a violent bout of cough. With a hypersensitive carotid sinus, stooping, bending or turning of the head or a tight collar may cause giddiness.

(3) *Premontory symptoms* Weakness, nausea, sweating and pallor accompany simple faints Usually, no premonitory symptoms in cardiac syncope A gradual development of syncope with dizziness, numbness and tingling of hands and feet is common in neurosis

(4) *Position in which syncope occurs* The patient may become light headed and giddy when first getting up in the morning This is suggestive of orthostatic hypotension

(5) *History of head injury* may be obtained in case of concussion

(6) *History of blood loss*

(7) *Use of drugs* Drugs likely to induce syncope are nitrites, ganglion-blockers and insulin It may also result from drug sensitivity to procaine or penicillin

(8) *Associated features* Dyspnoea and palpitation common in cardiac cases, apprehension, mental confusion and excessive sweating in hypoglycaemia, visual disturbances or tetany in hyperventilation Association of syncope with memory defects, temporary hemiparesis, vertigo or tinnitus in an older person suggests presence of vertebro-basilar insufficiency

## THIRST

Excessive craving for water or fluids is referred to as *thirst* A proper water or fluid-balance within the body is an essential physiological requirement, dependent on (1) an adequate supply of fluids for all bodily requirements, and (2) a proper distribution of fluids within the tissues of the body and blood One of the major manifestations of water-imbalance within the body is thirst

*Mechanism* The sensation of "thirst" has been ascribed to (1) drying of the oral and pharyngeal mucous membranes with reduced secretion of saliva, because of inadequate bodily water content, and (2) altered composition of the blood

## CAUSES

(1) *Physiological* Hot or warm weather (with excessive sweating), occupation (e.g. stokers), physical exercise, foods rich in salt

(2) *Excessive loss of fluids* Vomiting, diarrhoea, cholera, polyuria, haemorrhage, shock, anasarca

(3) *Drugs* Belladonna, atropine

(4) *Gastrointestinal* Pyloric obstruction, carcinoma, gastric stasis

(5) *Local causes* Mouth-breathing, adenoids, parotitis, mumps, salivary gland disorders

## URINARY DISORDERS

### TYPES OF DISORDERS AND CAUSES

Many symptoms are related to the act of urination, each with its own characteristics and significance. The main types of abnormalities encountered are

#### Dysuria (Painful micturition)

##### *Local causes*

- (1) *Urethral* Acute urethritis, gonorrhoea, balanitis
- (2) *Prostatic* Acute gonococcal prostatitis, carcinoma
- (3) *Bladder diseases* Acute cystitis, bladder-stone
- (4) *Gynecological and rectal* Fibroids, carcinoma

*Neurological* rare vesical crisis of tabes dorsalis

#### Frequency of Urination

*Painful frequency* Inflammatory or neoplastic lesions of urethra, bladder, prostate, uterus or rectum

*Painless frequency* Polydipsia or excessive intake of fluids, diabetes mellitus or insipidus, tea, coffee, alcohol, beer, cold weather, anxiety state, crystals in urine, retention with overflow, chronic nephritis, drugs (e.g. diuretics), spinal cord lesions

#### Retention of Urine

(1) *Interference with nervous mechanism of bladder* Tabes dorsalis, Pott's paraplegia, multiple sclerosis

(2) *Bladder lesions* Blood clot, carcinoma, papilloma, calculi

(3) *Prostatic lesions* Prostatic enlargement, carcinoma, prostatitis, prostatic abscess

(4) *Urethral lesions* Stricture, calculi, tumours

Acute retention of urine may occur without previous warning or may occur in patients who have had symptoms of bladder outflow obstruction for some time before the acute episode. The symptoms are

*Hesitancy* The patient has to wait an appreciable period of time before being able to void

*Diminution of stream* The flow becomes a feeble stream

*Frequency* Especially at night when the patient is awakened from sleep on at least two occasions



**Urgency** The patient feels he must void without delay only to be faced with hesitancy when voiding

**Precipitancy** If the patient is unable to get to a suitable place, he wets himself

### Incontinence

(1) *Physiological* in infancy

(2) *Stress incontinence* in women

(3) *Cerebral lesions* Coma, epilepsy, mental confusion, psychosis Lesions of frontal lobe of brain and of fibres which subserve voluntary control, multiple sclerosis, intracranial tumor, cerebral arteriosclerosis

(4) *Spinal cord lesions* Spinal cord compression, myelopathies, trauma, multiple sclerosis

(5) *Malingering*

### Dribbling

(1) *Physiological* Common in elderly individuals during coughing, sneezing or straining

(2) *Local causes* Cystocoele, rectocoele, weak perineum, paralytic bladder, prostatic obstruction

**Oliguria (and Anuria)** Oliguria is that amount of urine output (400-500 ml/24 hours) below which the normal load of metabolic waste products cannot be excreted Anuria is complete absence of urine secretion

(1) *Pre-renal* (a) Associated with hypotension fluid loss, cardiogenic or septic shock (b) Without hypotension Congestive heart failure, renal arterial obstruction such as thrombosis or embolism, volume depletion, 'third space' losses e.g. peritonitis, intestinal obstruction

(2) *Renal* Acute glomerular nephritis, acute tubular necrosis, bilateral cortical necrosis, hypercalcaemic or urate nephropathy, end stage of chronic renal failure

(3) *Post-renal* (a) Obstructive uropathy e.g. prostatic or urethral obstruction or rarely bilateral ureteral obstruction (b) Renal vein thrombosis

**Polyuria** means an excessive output of dilute urine As a result the patient is thirsty and drinks more

**A. Transient polyuria** Diuretic drugs, spontaneous remission in nephrotic syndrome, diuretic effect of alcohol, caffeine (tea, coffee), vitamin B<sub>1</sub> in beriberi, proteins in famine oedema

### B *Persistent polyuria*

- (1) True (cranial) diabetes insipidus
- (2) Nephrogenic diabetes insipidus
- (3) Renal disease Inability to concentrate urine may lead to obligatory polyuria
  - (a) Chronic renal failure
  - (b) Hypercalcaemic nephropathy
  - (c) Hypokalaemic nephropathy
- (4) Compulsive water drinking (psychogenic polyuria)

### Nocturia or nocturnal frequency of urine.

- (1) *Psychogenic* Disturbance of sleep due to any cause may prompt a desire to micturate
- (2) *Irritative* Increased frequency with nocturia due to enlarged prostate, or bladder cystocele Associated symptoms of dysuria and stress incontinence
- (3) *Polyuria* In kidney disease the earliest loss of concentrating power will affect the kidney's special ability of concentrating urine at night

## VERTIGO

Vertigo is derived from the Latin word 'Vertere' meaning 'to turn'

Consciousness of disordered orientation of the body in space is usually referred to as *Vertigo* It is a subjective sensation of turning or rotation either of the patient or his surroundings or both Vertigo may present itself in one of three ways (1) The body or head may be felt to rotate or move (2) The surroundings may display rotation or movement, or (3) The posture or movements of the lower limbs may appear unsteady Dizziness or a sensation of mere unsteadiness, as seen sometimes in cases of neurosis, head-injuries or cerebral atherosclerosis, not being associated with a sense of turning or falling, is not included under the caption of Vertigo

### CAUSES

- (1) *Aural*
  - (a) External ear e.g wax impacting on ear drum
  - (b) Middle ear e.g Eustachian tube block
  - (c) Internal ear Acute labyrinthitis, Meniere's syndrome
- (2) *Vestibular nerve* Vestibular neuronitis
- (3) *Cerebello-pontine angle* e.g acoustic neuroma, meningioma

- (4) *Brain stem* e.g. vertebro-basilar insufficiency, encephalitis
- (5) *Temporal lobe* e.g. trauma, neoplasm
- (6) *Ocular* Ocular muscle paralysis, car or train sickness, standing at unaccustomed heights
- (7) *Psychogenic* Anxiety neurosis, hysteria

#### METHOD OF INQUIRY

(1) *Paroxysmal or continuous* Vertigo may occur in episodes, with freedom from discomfort in between attacks, as in labyrinthitis. In Meniere's disease, it may be preceded by a sensation of uneasiness or discomfort lasting for a varying period of time. Persistent vertigo may follow vertebro-basilar infarction or be associated at times with chronic ear disease, also from streptomycin or kanamycin.

(2) *Disturbances of hearing* These, when present, are almost always caused by peripheral disturbances. Tinnitus, in the form of buzzing, ringing or whistling, is often encountered in otosclerosis, it may occur in Meniere's disease. It may also be produced by obstruction of the ear or Eustachian tube, or involvement of the auditory nerve.

(3) *History of trauma* Except for headaches, dizziness is the most common complaint following head injury.

(4) *Precipitating factors* Vertigo produced by head movement occurs in vertebro-basilar insufficiency, carotid sinus syncope and cervical spondylosis. Complaint of vertigo on lying down or sitting up in bed or on turning over (positional vertigo) may occur after head injury, following ear infections or after vestibular neuritis.

(5) *Associated symptoms* Nausea and vomiting, associated with severe vertigo are suggestive of Meniere's syndrome and thrombosis of the posterior inferior cerebellar artery. Attacks of tingling involving the extremities and mouth, may be noted in association with transient slurring of speech. In basilar artery insufficiency there may be a diplopia, facial dysaesthesia, dysarthria or the patient may fall suddenly because of transient loss of consciousness or through so-called "drop attacks", the patient falling suddenly without loss of consciousness and then regaining normal posture within a moment. Loss of consciousness may suggest temporal lobe epilepsy. Loss of consciousness is seldom if ever observed in labyrinthine vertigo or Meniere's syndrome.

## VISUAL LOSS OR FAILURE

Loss of vision in one or both eyes may be sudden and transient or may be due to chronic progressive visual loss

### CAUSES

Anatomically the lesion may be in

- (1) *Cornea* Opacities (cataract), scarring
- (2) *Aqueous* Pus or blood, acute glaucoma
- (3) *Lens* Cataract, refractive error
- (4) *Vitreous* Haemorrhage
- (5) *Retina* (a) *Congenital* Tay-Sach's amaurotic family idiocy, retinitis pigmentosa (b) *Acquired* Central retinal artery occlusion, central retinal vein thrombosis, retinal haemorrhage or detachment, diabetic retinopathy Spasm of retinal arteries due to methyl alcohol, quinine, tobacco
- (6) *Optic nerve, optic chiasma and its central connections* e.g. tabes, general paralysis, pituitary and other tumors, chronic arachnoiditis

## VOMITING (OR EMESIS)

By *vomiting* is meant a forceful expulsion of stomach contents through the mouth, as the result of increased intra-abdominal pressure, produced by abdominal and diaphragmatic contractions. The act of vomiting is frequently preceded or accompanied by nausea or a peculiar feeling of impending vomiting, usually experienced in the throat or epigastrium.

Due to a sudden rise of intra-abdominal pressure, secondary to simultaneous, vigorous and rhythmic contractions of the diaphragm and abdominal muscles, the gastrointestinal contents are forcefully expelled through the mouth, in vomiting. The act of vomiting is highly complex and involves descent and contraction of the diaphragm, spasm of abdominal muscles, pyloric spasm with relaxation of the cardia, forward and upward movement of the larynx and hyoid bone, elevation of the soft palate, closure of glottis and rise of intra-abdominal and intrapulmonary pressures.

The *mechanism* of vomiting is highly complex, including psychic, neurological and gastrointestinal components. The "*medullary emetic mechanism*" is comprised of a *vomiting centre* in the region of the fasciculus solitarius and a *chemoreceptor trigger zone* in the floor of the fourth ventricle. Whilst the latter is sensitive to emetic drugs (e.g. apomorphine, ergot, morphine, digitalis and copper sulfate), irradiation and motion-sickness, the former may be stimulated by impulses from the gastrointestinal tract or higher centres of the

brain Whilst *afferent* impulses from the alimentary tract travel via the sympathetic trunks, *efferent* ones are conveyed through the phrenic nerves to the diaphragm, spinal nerves to the intercostal and abdominal muscles and vagus nerves to the pharynx and larynx The *threshold* of vomiting varies greatly in individuals, being "low" in cases of functional vomiting

## CAUSES

Vomiting may be toxic, reflex, central or obstructive in origin The important causes of vomiting and nausea are

(1) *Gastrointestinal causes* Gastritis, cholecystitis, cirrhosis of liver, carcinoma of stomach, intestinal obstruction, virus hepatitis, pyloric stenosis, pancreatitis

(2) *Poisons* Food poisoning, alcoholism, uraemia.

(3) *Drugs* Digitalis, opium or morphia, anaesthetics, emetics, oestrogen, xanthines, antibiotics

(4) *Reflex causes* Migraine, ear disease, severe pain, Meniere's disease, sea- or air-sickness, colicky pains, labyrinthitis

(5) *Systemic disorder* Fevers, uraemia, pregnancy, congestive heart failure

(6) *Psychic causes* Unpleasant smells, tastes, fear, disgust or tragic sights, hysteria, neurasthenia.

(7) *Neurological causes* Cerebral tumour, meningitis, migraine, tabetic crises

(8) *Endocrine causes* Addison's disease, hyperparathyroidism, thyrotoxicosis

(9) *Habit vomiting* Vomiting initially occurs in a period of stress but it may persist long after the emotional stress has resolved

## METHOD OF INQUIRY

(1) *Time of onset* Vomiting occurring early in the morning before breakfast suggests a toxic cause, such as incipient uraemia or chronic gastritis (particularly due to alcohol) Vomiting occurring regularly and soon after food may be functional or hysterical in origin Vomiting of large amounts of well-digested food, at intervals of 12 to 24 hours or longer, is almost pathognomonic of pyloric obstruction.

(2) *Duration of complaint* A short duration suggests a toxic factor, poison, ingestion of some food or infective lesion, such as cholecystitis A long duration is common in hepatic cirrhosis, malignancy, Addison's disease

(3) *Ingestion of drugs, poisons or certain foods in the near past.*

(4) *History of similar episodes in the past* as in cyclical vomiting of children, hysteria or cholecystitis

(5) *Presence or absence of nausea* As a rule, vomiting secondary to common alimentary disorders is preceded by nausea. Vomiting without nausea suggests the possibility of an intracranial lesion, such as tumour

(6) *Pain* Vomiting which follows and relieves an episode of epigastric pain is usually due to an intragastric lesion or pylorospasm. In appendicitis or pancreatitis, vomiting may occur with abdominal pain. In biliary or renal colic, there is unilateral abdominal pain

(7) *Associated features* Periodic attacks of vomiting preceded by one-sided headache are diagnostic of migraine. Functional vomiting is more common in women, usually follows psychic trauma and is particularly common at the time of menstruation

(8) *Nature of vomit* This may be highly informative. Whilst a *faecal odour* suggests intestinal obstruction, gastrocolic fistula, peritonitis or long-standing pyloric or duodenal stenosis, *lack of odour* suggests achylia gastrica or achalasia. Presence of *undigested food* in the vomit, hours after eating, suggests pyloric stenosis, oesophageal or duodenal diverticulum, achylia gastrica or obstruction below the level of the common bile duct (when mixed with *bile*). Persistent vomiting of *mucus* suggests gastritis, pregnancy, malignancy or postnasal drip, of *pure gastric juice*, duodenal ulcer or gastric neurosis, of *bile*, obstruction below the ampulla of Vater, of *pus*, abscess or suppurative gastritis, of *blood*, a variety of conditions (described under Haematemesis), and of gall-stones, cholecysto-gastric or-duodenal fistula. Swallowed foreign bodies or intestinal worms may also be discharged via the mouth in vomiting

*Retching* is not synonymous with vomiting, indicating as it does violent rhythmic contractions of the respiratory muscles with a closed mouth. It may be followed by the act of vomiting

## WEIGHT LOSS (EMACIATION)

Underweight or a body weight below the normal average may be due to (1) *Thinness*, leanness or underdevelopment of fatty tissues. A defective gain in body weight or minor losses in weight may be due to factors, such as age, sex, race, familial predisposition and enforced dieting. (2) *Emaciation* or wasting, with abnormal or excessively rapid loss (or absence) of bodily fat, as in cases of malignancy and wasting disease. Emaciation associated with anaemia, pallor or a sallow tinge of skin, as in advanced cases of cancer or chronic congestive heart failure, is sometimes referred to as *cachexia*

The term *weight loss* refers to actual loss of body weight, which may be rapid or gradual. This is usually secondary to deficient ingestion or intake of food (as in starvation, worry, insomnia), poor assimilation or digestion of food, malabsorption of the end-products of digestion, defective metabolism or disease-states.

Emaciation, like obesity, may be exogenous or endogenous (or mixed) in origin.

## CAUSES

(1) *Deficient intake of food* Starvation, famine, anorexia, malnutrition, pain, worry, insomnia, fatigue

(2) *Dysphagia or difficulty in swallowing* Paralysis of tongue, palate or pharynx, cleft palate, hysteria, tetany, inflammation or tumour

(3) *Poor absorption* Prolonged vomiting, diarrhoea, pyloric obstruction

(4) *Gastric causes* Achylia, achlorhydria, gastric carcinoma

(5) *Intestinal causes* Chronic amoebiasis, ulcerative colitis, dysenteric infections, tuberculous enteritis

(6) *Pancreatic and hepatic diseases* Pancreatic tumour, chronic pancreatitis, hepatic cirrhosis

(7) *Cardiac causes* Chronic congestive heart failure, bacterial endocarditis

(8) *Neurological* Parkinsonism, advanced tabes, myopathies

(9) *Psychogenic* Anorexia nervosa, sexual neurasthenia

(10) *Endocrine types* Thyrotoxicosis, Addison's disease, Simmond's disease

(11) *Metabolic* Diabetes mellitus, uraemia

(12) *Avitaminosis* B Complex deficiencies

(13) *Traumatic and post-operative* Severe accidents, operations, burns (with acute negative nitrogen balance)

(14) *Toxic and infective diseases* Pulmonary tuberculosis

(15) *Haematological* Leukaemias, aplastic anaemia

(16) *Drugs* Alcoholism, opium habit, excessive smoking

## METHOD OF INQUIRY

(1) *Age* In children the commonest causes are malnutrition, injudicious feeding or gastrointestinal infections. In middle or old age malignancy should be suspected.

(2) *Sex* Anorexia nervosa occurs nearly always in females.

(3) *Degree of weight loss* Considerable weight loss may occur in pulmonary tuberculosis, malignancy or diabetes.

(4) *Dietary history* Study of the diet for deficient intake, either qualitative or quantitative

(5) *Appetite* Weight loss in spite of a good appetite suggests diabetes or thyrotoxicosis

(6) *Addiction* to drugs or alcoholism

(7) *Diarrhoea* Chronic diarrhoea would point to gastrointestinal infection or malabsorption syndrome as the likely cause

(8) *Fever, cough or haemoptysis* should suggest pulmonary tuberculosis

(9) *History of neurosis or depression* may be obtained from relatives

(10) *Hours of work, strenuous life, anxiety, insomnia and pain* should be inquired into



## 3 | Vital Data

AFTER completion of the history, the next step is a general inspection or observation of the patient "as a whole"—a sort of "bird's eye view" from head to foot. The discerning eye of a trained physician may obtain a wealth of useful information of great diagnostic value within a matter of a few seconds.

The temptation, however, for a spot diagnosis must be resisted. Although a spot diagnosis (snap diagnosis, instantaneous diagnosis) is possible in a large number of cases on observing some characteristic feature of the disease, such as the facial expression, set of the eyes or the gait, the final diagnosis must be withheld until the initial impression of the examiner is confirmed by a subsequent, more detailed examination.

**Prerequisites** There are certain rules which can be profitably observed during the initial inspection of the patient. The patient must always be examined in a good light, in a quiet room, and when completely relaxed and comfortable. He should be partially or completely undressed. Daylight is always preferable for a good examination, conditions such as anaemia, jaundice, and certain skin lesions are likely to be misinterpreted or even missed completely in artificial lighting.

The physician must be relaxed, comfortable, observant and inquisitive.

A systematic routine, involving careful observation of the patient from head to foot, is essential for diagnosis. Errors in diagnosis are often due to careless, haphazard or hasty inspection. Clinical diagnosis depends on four methods of examination, viz inspection, palpation, percussion and auscultation. The tendency on the part of some physicians to rely solely on auscultation for diagnosis of chest diseases can only be deprecated as it is likely to lead to serious errors of omission. For instance, a mitral stenotic lesion, which is quite obvious on palpation because of a characteristic presystolic thrill, may be easily missed through hasty or improper auscultation.

## RECORD OF PHYSICAL EXAMINATION

## PRELIMINARY DATA

Name	Male or Female	Age	Race	Date
Address		Occupation		

## VITAL DATA

<i>A</i> Temperature	<i>B</i> Pulse	<i>C</i> Respiratory rate	<i>D</i> Weight	<i>E</i> Height
<i>F</i> Blood pressure	<div style="display: inline-block; text-align: center;">           Systolic            _____            Diastolic         </div>		Pulse pressure	

## GENERAL OBSERVATIONS

<i>A</i> Constitution	<i>B</i> Stature	<i>C</i> State of nutrition	<i>D</i> Posture	<i>E</i> Emotional state
<i>F</i> The Skin	<i>G</i> The Hair			

## HEAD

<i>A</i> Skull or cranium (size, shape, fontanelles, sutures)	<i>B</i> Hair	<i>C</i> Movements of head
<i>D</i> Forehead	<i>E</i> Face (type, colour, spasm)	

## EYES

<i>A</i> Expression	<i>B</i> Eyebrows	<i>C</i> Eyelids	<i>D</i> Palpebral fissure	<i>E</i> Lachrymation
<i>F</i> Eyeballs	<i>G</i> Tension	<i>H</i> Squint	<i>I</i> Nystagmus	<i>J</i> Conjunctivae
<i>L</i> Cornea	<i>M</i> Iris	<i>N</i> Pupils		

## EARS

<i>A</i> Appearance	<i>B</i> Discharge	<i>C</i> Deafness	<i>D</i> Lesions
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## NOSE

<i>A</i> Appearance	<i>B</i> Discharge	<i>C</i> Nostrils	<i>D</i> Sense of smell
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## MOUTH AND THROAT

<i>A</i> Lips (colour, lesions, twitching, herpes, smile)
<i>B</i> Breath
<i>C</i> Tongue (appearance, colour, furring, size, shape, fissuring, movements, lesions, ulcers, atrophy, deviation)
<i>D</i> Teeth (cavities, caries, pyorrhoea, colour, missing teeth, dentures)
<i>E</i> Gums (colour, pigmentation, swelling, ulceration)
<i>F</i> Buccal mucosa (inflammation, ulcers, colour)
<i>G</i> Palate (hard and soft palate)
<i>H</i> Floor of mouth
<i>I</i> Tonsils (size, follicles)

## NECK

- A* General appearance
- B* Trachea
- C* Lymphnodes (different groups of cervical glands)
- D* Thyroid gland (size, shape, goitre, bruit)
- E* Salivary glands
- F* Cysts and tumours
- G* Blood vessels

## UPPER EXTREMITIES

- A* Deformities and contractures
- B* Local swellings
- C* Oedema
- D* Muscles,
- E* Lymphnodes
- F* Joints
- G* Bones
- H* Blood vessels
- I* Hand (shape, deformities type, temperature, moisture, colour, handshake, handwriting, outstretched hand, abnormal movements)
- J* Fingers
- K* Nails (shape, appearance, lesions)

## LOWER EXTREMITIES

- A* Deformities and contractures
- B* Local swellings
- C* Oedema
- D* Lymphnodes
- E* Joints
- F* Bones
- G* Blood vessels
- H* Toes
- I* Nails

## BREASTS

Size, shape, symmetry, lumps, scarring, nipples, secretion, areola, discharge, ulceration, pigmentation, lymphnodes

## BACK

Spine, curvatures, symmetry, angulation, movements or mobility, tenderness

## GENITALS

Male genitalia, female genitalia

## CARDIOVASCULAR SYSTEM

- A Inspection* Size, shape and type of chest, shape of precordium Apical thrust (displacement, extent, site, force, absence, type) Other precordial pulsations Other thoracic and extrathoracic pulsations Arterial and venous neck pulsations Enlarged vessels Epigastric pulsations
- B Palpation* Apical thrust (site, extent, force, character, type, mobility) Other pulsations Thrills (site, timing, extent) Pericardial rub Palpable sounds
- C Percussion* (cardiac borders, cardiac area of dullness, supra-cardiac dullness)
- D Auscultation* Heart sounds (rhythm, rate, intensity and abnormalities of first sound, intensity, splitting and abnormalities of second sound, systolic extra sounds, diastolic extra sounds) Murmurs (timing, site, extent, intensity, transmission, duration, quality, pitch, thrill, effects of posture, respiration and exercise) Friction sounds
- E Pulse* Rate, rhythm, vessel wall, volume, force, tension, equality, contour, type

## RESPIRATORY SYSTEM

- A Inspection* Size, shape, type, symmetry, respiratory expansion, equality of movement, type of breathing, rate and rhythm
- B Palpation* Confirm expansion, local swellings, local tenderness, tactile vocal fremitus
- C Percussion* Topographical percussion, apical, basal and midzone, Kronig's isthmus, axillary percussion, myotatic irritability
- D Auscultation* Breath sounds (type, character, intensity, length of phase) Adventitious sounds (type, site, extent, timing) Friction rub (site, extent, characteristics, timing)

## ABDOMEN

- A Inspection* Contour or shape, symmetry, local bulgings or retractions, state of skin, umbilicus, recti muscles, blood vessels, peristalsis, respiratory movements
- B Palpation* Rigidity and feel, oedema of wall, tenderness, investigation of lump (if any) palpability of liver, spleen, gall-bladder, kidneys, stomach, pancreas, small intestine, colon and urinary bladder, palpation of hernial orifices
- C Percussion* Tympanitis, signs of ascites, liver percussion, percussion of other organs
- D Auscultation* Peristaltic sounds and other sounds
- E Abdominal mensuration*
- F Rectal examination*

## NERVOUS SYSTEM

- A Higher function* Intelligence, memory, delusions or hallucinations, state of consciousness
- B Speech* Understanding and expression of spoken and written speech, type of aphasia or dysarthria (if any)
- C Cranial nerves* Test each cranial nerve (systematically), sense of smell, visual acuity, visual field, colour vision, fundi, eyeball movements, squint, nystagmus, pupils, pupillary reflexes, masseters, temporal muscles, pterygoids, facial movements, skin of face, hearing, balance, sensation of tongue, palatal and pharyngeal reflexes, shrugging of shoulders, sternomastoids, tongue wasting and deviation
- D Motor system* Loss of muscle power of any important muscles of the body, increase or decrease and type of muscle tone, coordination of muscles and state of nutrition, wasting or hypertrophy, abnormal muscle movements
- E Sensory system* Exteroceptive and proprioceptive sensations and cortical sensory function
- F Reflexes* Superficial, deep and visceral reflexes
- G Cranium and spine*
- H Gait* Type of gait (if abnormal)

## TEMPERATURE

A rough idea of the temperature of the body may be gained by laying the palm of the hand over the patient's bare body, provided the skin be dry. For practical purposes, however, such a method is both crude and inaccurate. It is therefore incumbent on the physician to use, for this purpose, a clinical

thermometer, with a Fahrenheit or Centigrade scale, the latter being favoured by the more scientifically minded. For accurate thermometry, (1) the thermometer should be of reliable make and certified as accurate, (2) it should be kept in position for at least two to three minutes, (3) the temperature may be recorded or observed in the mouth (oral), axilla (axillary), groin (inginal) as in infancy and early childhood, or rectum (rectal), (4) for oral registration, the mouth must be kept closed whilst breathing proceeds through the nose, (5) for axillary recording, the skin must not be chilled or moist with perspiration, (6) the mercury must be well shaken down before using the thermometer (but after washing), (7) in case of pyrexia, the temperature should be recorded every four or six hours in order to determine the type of fever.

**Normal temperature** In warm-blooded animals, mammals and man, a constant body temperature is maintained, irrespective of the atmospheric temperature. In man, the normal temperature in the mouth is  $98.6^{\circ}\text{F}$  ( $37^{\circ}\text{C}$ ) with a range from  $97^{\circ}\text{F}$  ( $36^{\circ}\text{C}$ ) to  $99^{\circ}\text{F}$  ( $37^{\circ}\text{C}$ ), in the axilla  $98^{\circ}\text{F}$  ( $36.5^{\circ}\text{C}$ ) with a range from  $96.5^{\circ}\text{F}$  ( $35.5^{\circ}\text{C}$ ) to  $98.6^{\circ}\text{F}$  ( $37.0^{\circ}\text{C}$ ), and in the rectum  $99.2^{\circ}\text{F}$  ( $37.3^{\circ}\text{C}$ ) with a range from  $97.5^{\circ}\text{F}$  ( $36.3^{\circ}\text{C}$ ) to  $98.8^{\circ}\text{F}$  ( $37.7^{\circ}\text{C}$ ). Normally, the temperature regulation of the body is very efficient, particularly in women.

**Abnormal temperatures** In states of disease, the heat regulating mechanism may become deranged and the bodily temperature may be deviated (to a greater or less extent) from normal. Any temperature of over  $99^{\circ}\text{F}$  in the mouth is usually referred to as *fever* or *pyrexia*, whilst a temperature of over  $106^{\circ}\text{F}$  constitutes hyperpyrexia (danger zone of pyrexia).

**Types of fever** (Fig 3.1) The main types of fever are continuous, intermittent, remittent, periodic and irregular.

When in case of fever the temperature does not touch normal at all, it is either continuous or remittent. If the diurnal fluctuations exceed  $1\frac{1}{2}^{\circ}\text{F}$  in 24 hours, the fever is termed *remittent*, whilst with fluctuations of less than  $1\frac{1}{2}^{\circ}\text{F}$  per day, the fever is called *continuous*. A fever that touches normal for a few hours during the day is called *intermittent*, *hectic* or *septic*, intermittent fever that occurs daily is termed *quotidian*, when on alternate days, *tertian*, and when every fourth day, *quartan*. A fever that does not conform to any of these three types is called *irregular* fever. A fever occurring in bouts lasting for days with afebrile phases is called relapsing or periodic fever. Relapsing type of pyrexia may occur in brucellosis, Hodgkin's disease and spirochaetal relapsing fever.

**Stages of fever** In the course of a fever, the following stages are usually recognizable: (1) the prodromal stage (which may be absent), (2) the stage of onset or invasion (initial or pyrogenetic phase), (3) the stage of full development (fastigium), (4) the stage of decline (defervescence or termination).

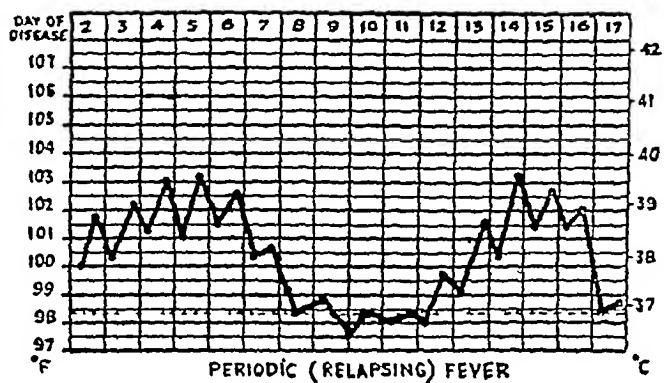
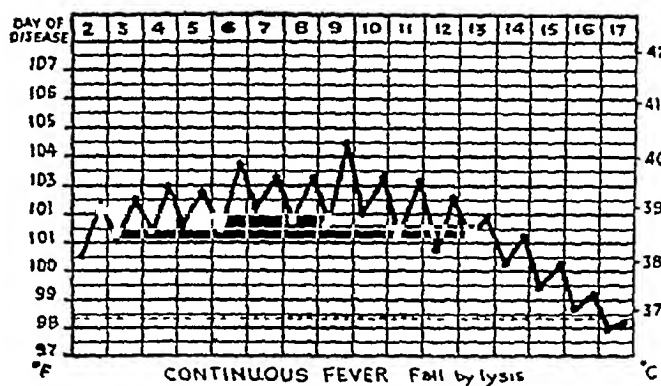
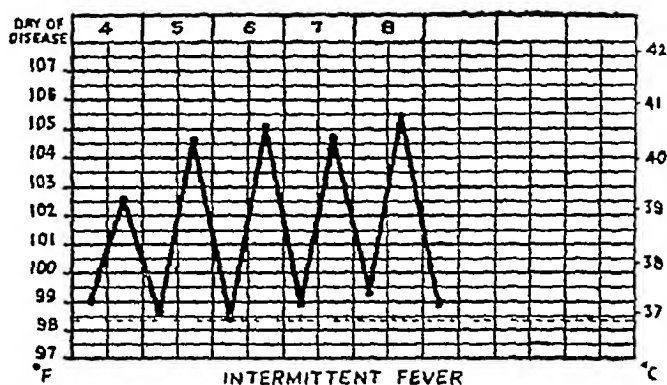
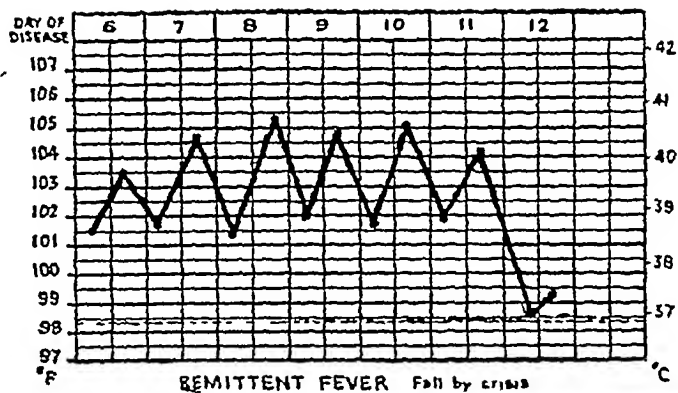


Fig. 31 Common types of fevers

The onset of a fever may be insidious (gradual) or at times of step-ladder type, as in typhoid fever, or acute (abrupt) or sudden as in lobar pneumonia or small-pox. The termination or fall of a fever may be sudden (by *crisis*) as in pneumonia, or gradual (by *lysis*) as in typhoid fever. A transitory fall to normal with subsequent rise of temperature is called pseudocrisis.

**Accompaniments** A sudden rise of temperature may be accompanied by a rigor or chill as in adults, or by *convulsions* as in children. Chills or rigors are commonly observed in pyelonephritis, lobar pneumonia, malaria, septic fever, bacterial endocarditis. Recurrent rigors can occur in septicaemia, pyaemia and bile duct or liver sepsis. A sudden and marked drop of temperature or severe crisis may lead to collapse, delirium or even death.

**Mechanism of fever** Fever is a symptom and not a disease. The main mechanisms concerned in the causation of fever are (1) increased production of heat, as in infectious fevers and postoperative pyrexia, (2) decreased dissipation of heat, as in heat stroke or severe dehydration or (3) a combination of both mechanisms, as in malignancy, blood dyscrasia or sunstroke.

**Subnormal temperatures** A temperature below 97° F by mouth may be termed subnormal. A sudden drop of temperature to subnormal may arise after injury, operation, burns, haemorrhage, acute illness or shock. A persistently low temperature is common in emaciation, or wasting diseases, myxoedema and Simmonds disease.

**Clinical causes** The major causes of pyrexia are (1) Infections. Bacterial, viral or rickettsial, spirochaetal, protozoal, fungal. (2) Malignant disease. (3) Allergic disorders. (4) Collagen diseases. (5) Collection of pus. (6) Heat fever. (7) Factitious fever.

## RATE OF THE PULSE

The pulse is counted, after resting the patient for a few minutes to allow complete relaxation, at the wrist for at least one minute and the rate is recorded as X beats per minute, the first beat is not counted.

**Normal rate** The rate varies from patient to patient and even in the same patient from time to time. In adults, the rate per minute may range from 60 to 90, although usually between 70 and 75. The rate is somewhat higher in children and women and is increased by standing, eating, exercise, and emotional factors.

The pulse rate may be unduly high during fever, in diseases like thyrotoxicosis, infectious diseases such as streptococcal infection, diphtheria, and septic fever, paroxysmal tachycardia (where the rate is usually over 160 per minute, attaining at times rate of even 300 or more per minute), and after drugs such as atropine and adrenalin.

**Abnormal rate** A pulse rate of over 100 per minute, called *tachycardia* is considered abnormal and requires investigation, provided emotional strain and physical effort are not responsible for the same.

A pulse rate below 60 beats per minute is called *bradycardia*. Although, at times, a familial or hereditary characteristic and common enough in athletic individuals in training (even as low as 40 per minute), a slow pulse may be indicative of certain clinical states, particularly (1) convalescence after infectious diseases like pneumonia and influenza, (2) during the course of enteric or typhoid fever, (3) jaundice, (4) raised intracranial tension, and (5) heart block. In complete heart block, the rate may be 30 to 40 beats per minute and regular. In rare cases, ventricular rates of even nine and ten per minute have been recorded.

Some individuals display a capacity to voluntarily influence their pulse rates. Such a voluntary control of the pulse rate has been attributed to vagus or sympathetic effect. A drop of rate of this kind from 76 to 24 per minute, was witnessed recently in a yoga. Voluntary or volitional obliteration of the pulse at the radial artery is due to pressure of the scalenus group of muscles on the subclavian artery and not to cardiac arrest. A stoppage of the pulse of 30 minutes duration was displayed by Coll Townsend in London. Voluntary acceleration of the pulse is rare and due to abnormal sympathetic control.

## RESPIRATORY RATE AND RHYTHM

### NORMAL RESPIRATION

In health, the *rate* of respiration in adults is about 16 to 20 per minute, being somewhat higher in female subjects. The respiratory rate is higher in childhood and infancy, being 40 or over per minute at the time of birth. The rate of respiration is greatly affected, even in health, by exertion, excitement and emotion. The *rhythm* of respiration is normally regular, but may become irregular during excitement, emotion or self-consciousness, and periodic during sleep in old age.

Normal *inspiration* is an active process dependent on muscular action (intercostal muscles, diaphragm and abdominal muscles) and associated with an upward and outward movement of the ribs resulting in expansion and upward movement of the thorax. Normal *expiration*, slightly longer in duration than inspiration, on the other hand, is a passive process associated with an inward and downward movement of the ribs and a falling-in of the chest wall.

The *respiration-pulse ratio* first described in ancient China many centuries before Christ, is normally about 1 to 4 (18 respirations to about 72 pulse beats per minute).

The *type of breathing*, in health, depends greatly on the sex and age of the patient. In women, the intercostal muscles play a dominant role in respiration, the respiratory movements mainly affect the upper part of the thorax, and the type of breathing may be described as thoracic, thoracico-abdominal or costal. In men and children, the respiration is mostly abdominal and dependent on the diaphragm, the type of breathing being described as abdominal, abdomino-thoracic or diaphragmatic.

Normally, the two sides of the chest move equally or symmetrically and simultaneously during respiration, the expansile and upward movement of the chest wall being associated with a mild degree of inspiratory retraction of the lower intercostal spaces and a symmetrical outward movement of the subcostal margins on the two sides.



## ABNORMAL RESPIRATION

**Abnormal rate** An increase in respiratory rate may be either physiological (during excitement or exertion) or pathological (as in fever and diseases of the lungs and heart). It may be compensatory as in the case of the painful shallow breathing of pleurisy or peritonitis. Increased rate of respiration is referred to as polypnoea or tachypnoea. A decrease in respiratory rate is common in cases of narcotic poisoning (e.g. morphia or opium), endocrine diseases associated with hypometabolism, uraemia, diabetic coma and raised intracranial tension.

**Abnormal rhythm** (Fig 3.2) The regular rhythm of normal respiration may be disturbed under various conditions, e.g. during excitement or emotion.

- (1) *Irregular respiratory rhythm* may be noted in meningitis, coma, peripheral circulatory failure or as preterminal event in moribund patients.
- (2) *Periodic breathing* is a special variety of respiratory arrhythmia or disorder where paroxysms of hyperpnoea or deep breathing alternate with phases of apnoea or cessation of breathing. Two main varieties of periodic breathing are recognizable, viz. (a) *Cheyne-Stokes breathing*, a fairly common variety of breathing associated with deep sleep, narcotic poisoning, or with cardiovascular, respiratory, renal and intracranial diseases, and characterized by a gradual waxing and waning of respiratory movements during the phases of hyperpnoea. Prolonged apnoea after hyperventilation is another manifestation of lesions of the cerebral cortex. (b) *Biot's breathing*, a rare type of periodic breathing usually associated with meningitis and characterized by sudden increase and decrease of respiration during the hyperpnoeic paroxysms.
- (3) *Suspirous breathing* (sighing respiration) is a special variety of respiratory arrhythmia usually associated with states of nervous excitement, fatigue, or neurocirculatory asthenia and characterized by interruption of normal respiration by occasional long and deep sighing breaths.
- (4) *Apneustic breathing* is characterised by a slow respiratory rate with respiration stopping in the maximal inspiration and is seen with mid-pontine lesions. Apneustic breathing is often referred to as 'inspiratory cramp'.
- (5) *Ataxic breathing* With medullary lesions irregularities of rate and amplitude may be observed. The respiration consists of series of irregular gasps or short inspiratory efforts terminating abruptly.

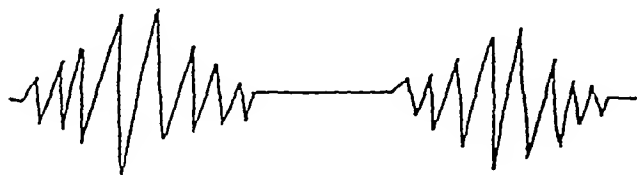
**Prolongation of respiratory phase** The inspiratory or expiratory phase (or both) may display undue prolongation in disease. Obstructive lesions of the upper air passages, particularly of the glottis and larynx (e.g. laryngeal diphtheria or glottic spasm) are liable to cause undue prolongation of the *inspiratory phase* of respiration, frequently associated with noisy or stridulous breathing. In obstructive lesions below the level of the larynx and particularly in bronchial asthma and emphysema, the *expiratory phase* is unduly prolonged and "wheezy" giving rise to the so-called asthmatic breathing.



NORMAL



HYPERVENTILATION



CHEYNE - STOKES



SIGHING



BIOT'S BREATHING



APNEUSTIC BREATHING



ATAXIC BREATHING

Fig. 32 Respiratory rhythms, normal and abnormal

Prolongation of *both phases* may be noted in acute alcoholic or narcotic poisoning, diabetic coma, uraemia or cerebral tumour, giving rise to the so-called slow breathing or *Kussmaul breathing*

**Altered respiration-pulse ratio** The normal respiration-pulse ratio of 1 to 4 may be altered in disease. In cases of pneumonia and at times in congestive cardiac failure (especially when associated with heart block) the ratio may be 1 to 3 or 1 to 2, in rare cases, the ratio may be even 1 to 1. The exact opposite may be noted in cases of opium or morphia poisoning, where the ratio may be 1 to 5 or 1 to 6.

**Abnormal type of breathing** An abdominal or male type of breathing in a woman is suggestive of thoracic disease, such as acute pleurisy, malignancy or arthritis of the dorsal spine. A costal or female type of respiration in a man or child may suggest the possibility of intra-abdominal diseases, such as ascites, massive hepatomegaly or splenomegaly, large tumour, peritonitis or diaphragmatic pleurisy.

*Exaggeration* of the normal type of respiration may also be indicative of disease. For instance, an entirely thoracic type of respiration (instead of the normal thoracico-abdominal type) in a woman is suggestive of intra-abdominal disease or diaphragmatic paralysis, similarly, an entirely abdominal or diaphragmatic type of breathing in a man or child suggests the possibility of dry pleurisy or intercostal muscle paralysis.

#### TYPES OF DYSPNOEA

Several varieties of dyspnoea are recognizable (1) *Exertional dyspnoea* or dyspnoea of effort, coming on after physical exertion. (2) *Paroxysmal* and nocturnal, mostly due to left ventricular failure or pulmonary oedema. (3) *Continuous dyspnoea*, an advanced form of dyspnoea, the laboured breathing continuing throughout the twenty-four hours. (4) *Orthopnoea*, a variety of postural dyspnoea, that is worse in the recumbent and relieved by the sitting, standing or semi-sitting position. (5) *Trepopnoea*, another variety of postural dyspnoea, relieved or exaggerated by one or other lateral position. (6) *Periodic respiration*, where phases of heavy breathing alternate with apnoeic pauses of no breathing. Two varieties of periodic breathing may be seen. In *Cheyne-Stokes* respiration, there is a gradual waxing and waning with alternating phases of hyperpnoea and apnoea. In *Biot's* respiration the changes are abrupt or sudden. (7) *Inspiratory dyspnoea*, maximal during the phase of inspiration. (8) *Expiratory dyspnoea*, during expiration. (9) *Kussmaul breathing*, noisy and slow breathing, as in diabetic coma or uraemia. (10) *Shallow and jerky breathing*, as in dry pleurisy or pneumonia with pain.

# 4 | General Observations

## CONSTITUTION

THE constitution (*diathesis, habitus*) or body-type of the individual may have a bearing on the disease process. On the basis of bodily configuration it is possible to classify the human race into certain basic constitutional or somatic types, the so-called *somatotypes*.

### CLINICAL CLASSIFICATION

The simplest and most useful method of somatotyping is based on simple clinical observation of the bodily habitus (Fig 4 1)

(1) **Asthenic (or hyposthenic)**, thin, long and underdeveloped body with long neck, flat chest, prominent hypogastrium, slender fingers and with a proclivity to neurasthenia and visceroptosis (Longilinear somatic constitution with vertical heart)

(2) **Normosthenic (or orthosthenic)**, with a normal average body build

(3) **Sthenic (or hypersthenic)**, broad, short and fat with short neck, muscular chest, and large and stumpy fingers (Brevilinear somatic constitution with horizontal heart)

(4) **Phthisical or phthinoid** (tuberculous diathesis), an exaggeration of the hyposthenic type, with flat chest, winged scapulae, lean body, poorly developed musculature and with a proneness to pulmonary tuberculosis

(5) **Plethoric or apoplectic**, an exaggeration of the hypersthenic type with flushed face or florid complexion, protuberant abdomen and with a tendency to cardio-vascular or cerebro-vascular accidents and emphysema

(6) **Athletic**, a variety of the hypersthenic type with overdeveloped musculature and broad chest

(7) **Endocrine** In the male type of endocrine habitus, the individual is excessively hairy and virile, whilst in the female type, there is less of hair, a delicate skin and fatty breasts and hips

(8) **Dysplastic**, or "mixed" constitutional type, where two or more of the above types are blended

## ANTHROPOMETRIC CLASSIFICATION

On the basis of bodily measurements or physical anthropometry, it is possible to distinguish three main types of humans (according to the preponderance of one or other of the three primary germ layers) (1) *Endomorph*, with soft, round contours and well developed cutaneous tissues (2) *Mesomorph* Wide, stocky, muscular individual (3) *Ectomorph*, with long narrow hands and feet, long, frequently shallow thorax and a small waist. Although scientific, this method of somatotyping is time-consuming and inconvenient

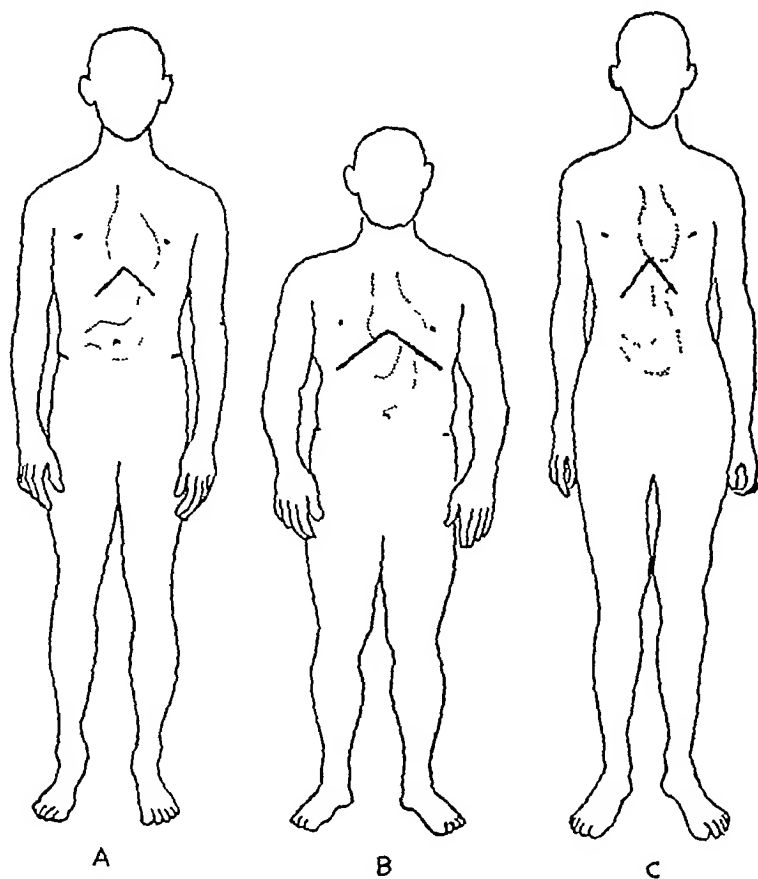


Fig 41 Types of body build A. Normosthenic, B Hypertrophic, C Hyposthenic

## AUTONOMIC CLASSIFICATION

On the basis of relative preponderance of the sympathetic or the parasympathetic system, two main types of individuals can be recognized (1) the *sympatheticotonic* type (lively, exuberant and active with warm skin, bright eyes and rapid heart) and (2) *vagotonic* (parasympatheticotonic or cholinergic) type (reserved and quiet, with pale and sweaty skin, small pupils, slow heart rate and low blood pressure). This method of typing has been more or less abandoned of late

## STATURE

On the basis of stature or total height from the top of the head to the soles of the feet, individuals can be classed as normal, tall or short. The term giant or dwarf is applied to an individual with an abnormally large or small stature.

The stature is to some extent dependent on race (cf., the tall Swede or Pathan and the short African Pigmy or the Gurkha), heredo-familial factors and sex, males being as a rule somewhat taller than females.

**Upper and lower measurements** The *stature* of the individual is the sum-total of the upper measurement (from the top of the head to the upper border of the symphysis pubis) and lower measurement (from the top of the symphysis pubis to the soles of the feet). The *span* is the distance between the tips of the fingers of the two hands, with the arms held horizontally outwards from the body.

Normally, the span and stature are about equal and so are the upper and lower measurements. In the event of premature or early epiphyseal union as in an adrenal cortex tumour with precocious puberty, the stature is greater than the span and the upper measurement greater than the lower. In delayed or retarded epiphyseal union as in hypogonadism or eunuchoidism and in certain other endocrine disorders, the span is greater than the stature and the lower measurement greater than the upper. In Marfan's syndrome the classical findings are long extremities with an arm span greater than the height (dolichostenomalia) the upper segment (head to pubis) to lower segment (pubis to feet) ratio being less than 0.85.

## GIGANTISM

Gigantism (giantism or macrosomia) is a term usually applied to a generalized and symmetrical overgrowth of the individual with increased skeletal length. When the height of an individual is far in excess of the average normal for his age and race, the individual is referred to as a giant. The term is frequently applied to any individual exceeding a height of 6 ft 6 in. Amongst the tallest individuals on record may be mentioned the Russian giant Machnow, who was 9 ft 3 in tall.

## FORMS OF GIGANTISM

**True gigantism** (Fig. 4.2) The usual form of gigantism, affecting the body as a whole, is referred to as true or generalized gigantism. This may be either hereditary or endocrine.

**Hereditary** (primary, genetic or simple) gigantism where in spite of the statural overgrowth the body is perfectly proportioned and the individual normal mentally, physically and sexually.

**Endocrine gigantism** Since statural growth is normally under the control of the eosinophil cells of the anterior pituitary gland, endocrine gigantism is almost invariably caused by *hyperpituitarism*. This may be *primary*, due to adenoma or hyperfunctioning of the anterior pituitary gland itself, or *secondary* due to endocrine involvement elsewhere, e.g. affections or tumours involving the gonads, pineal gland, adrenal cortex or floor of the third ventricle. The two common varieties of endocrine giants are the hyperpituitary giant and the eunuchoid giant.

**Hyperpituitary gigantism** Hyperfunctioning of the anterior pituitary gland, when established early in life prior to the fusion or union of the epiphyses, results in gigantism, when delayed till after epiphyseal fusion, it results in acromegaly. The hyperpituitary giant is usually well proportioned and frequently displays splanchnomegaly, good sexual development, symptoms of increased intracranial tension and polyuria or glycosuria.

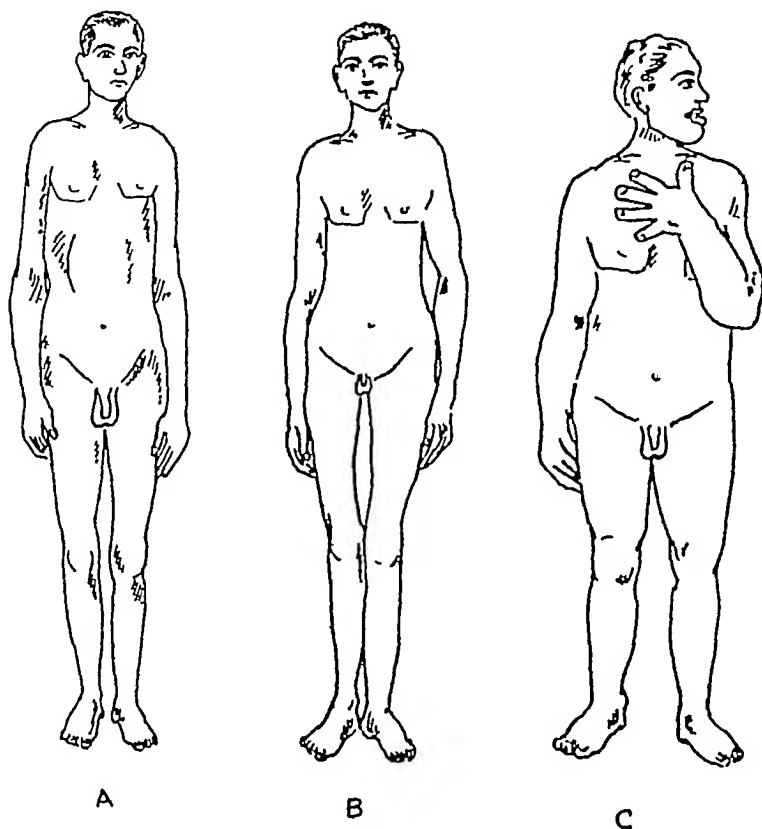


Fig 4.2 A True giant B Eunuchoid giant C Acromegalic giant

*Eunuchoid gigantism* The eunuchoid or hypogonadal giant is characteristically a tall, lanky and long-limbed individual (the lower limbs being particularly long) with a childlike voice, wizened face and infantile sex organs

Among special forms of gigantism are (1) *foetal gigantism*, or excessive statural growth in infancy or in the newborn, and (2) *localized gigantism* or overgrowth confined to local segments or parts of the body, as in congenital macroglossia, hemihypertrophy of face or limbs and macrodactyly (Fig 4 3) Overgrowth of an extremity or digit (macrodactyly) may be due to congenital peripheral arteriovenous fistula

## DWARFISM

Dwarfism or dwarfishness is a term applied to a shortness of stature or bodily development, irrespective of mental, muscular and sexual development An abnormally short individual is called a dwarf

The term *infantilism* must be clearly differentiated from dwarfism Infantilism or juvenilism refers to a uniform retardation or delay of development, inclusive of statural, muscular, mental and sexual development There is a persistence in adult life of the various characteristics of infancy or childhood (Fig 4 4) Whereas infantilism is nearly always associated with dwarfism, dwarfism more often than not exists without infantilism

Dwarfs have figured prominently in the art and literature of bygone days and continue to attract crowds in circuses and freak shows Amongst the shortest dwarfs on record may be mentioned the 2 ft. 7 in tall Charles Stratton (Tom Thumb) of Barnum Circus fame and the Indian dwarf, Jaggat Singh

## FORMS OF DWARFISM

**Hereditary (genetic or primary) dwarfism** A short stature may be an inherited characteristic as in *racial* dwarfism, for example African pygmies, Lapps and Gurkhas, *familial* dwarfism, and *sporadic* dwarfism where all the other members of the family are of normal stature The hereditary dwarf, except for his short stature, is normal in all respects

**Low birth weight.** A large group arises from children who had a low birth weight, these can be divided into three groups (a) Those who were born before their time—premature infants (b) Those who have suffered intra-uterine growth retardation secondary either to maternal disease or poor intrauterine environment. These children have characteristic features at birth namely marasmus, dry skin, wizened facies (c) Children of low birth weight who often have dysmorphic features including odd facies, minor congenital malformations and, in about half the cases, mild mental retardation

**Endocrine dwarfism.** Depending on the endocrine gland or glands affected the following varieties are known



*Cretinism* The cretin or hypothyroid dwarf displays a short stature, large head, mental retardation, coarse dry and unruly hair, swollen or puffy eyelids, flattened nose, thick lip, protruding tongue, coarse dry skin, spade-like hands and feet, short and bowed legs, infantile genitalia, curvature of spine, a protuberant abdomen with herniation, excessive sluggishness and stupid expression (Fig. 4 5)

*Fröhlich's syndrome* Usually ascribed to hypothalamic or pituitary dysfunction, Fröhlich's syndrome or dystrophia adiposogenitalis is characterized by a short stature, obesity, genital underdevelopment and normal intelligence (Fig. 4 6)

*Pituitary dwarfism* The pituitary dwarf (Fig. 4 7), although normal at birth displays a slow rate of growth and develops into a characteristically short-limbed individual with lack of axillary and pubic hair, sexual underdevelopment, immature childlike features, normal intelligence and easy fatiguability. This variety of dwarfism is always associated with infantilism.

*Gonadal dwarfism* A short stature, excessively long limbs, sexual underdevelopment and a scanty growth of hair in the axillary and pubic regions are characteristic of gonadal or hypogonadal dwarfism or infantilism (Fig. 4 8)

*Precocious sexual maturity* (*Macrogenitosomia praecox*) (Fig. 4 9) Although in early life, there is normal or rapid bodily growth with sexual precocity, premature fusion of epiphyses leads to delayed dwarfism later in life.

*Primordial dwarfism* Because of selective deficiency of the growth hormone, the adult is a miniature replica or "pocket-book edition" of a normal person. The pygmies of central Africa and genetic dwarfs are examples of this type, although hypogonadism is sometimes superimposed in genetic dwarfism.

*Polyostotic fibrous dysplasia* (*Albright's syndrome*) A disease of females with isolated cysts within the bones, short extremities (because of premature release of gonadotropins) and café-au-lait spots.

*Gonadal dysgenesis* (*Turner's syndrome*) Diminutive female with webbing of neck, gonadal dysgenesis, cubitus valgus, lymphoedema and coarctation of aorta.

*Disostosis multiplex* (*Hurler's syndrome*) Dwarfism associated with a large and grotesque head, protuberant abdomen, mental retardation, loss of vision and hepatosplenomegaly.

*Chondrodystrophy* (*Marquis's syndrome*) Disproportionate shortening of spine and extremities, resulting in small arms and legs, marked kyphosis, pigeon-breast, short neck and a waddling gait.

*Skeletal dwarfism.* This may be due to (1) *developmental* causes, such as achondroplasia, osteogenesis imperfecta, chondro-osteodystrophy, dyschondroplasia and gargoylism, or (2) *acquired* diseases, such as rickets, Pott's disease and spinal poliomyelitis.

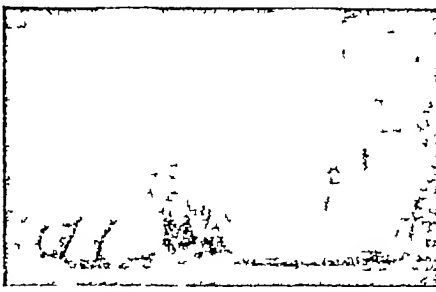


Fig 4.3 Localized gigantism affecting toes

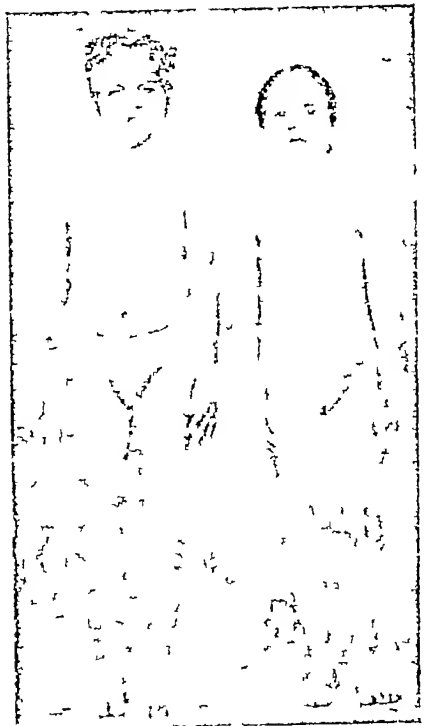


Fig 44 Pituitary infantilism in brother and sister aged 23 and 22 years

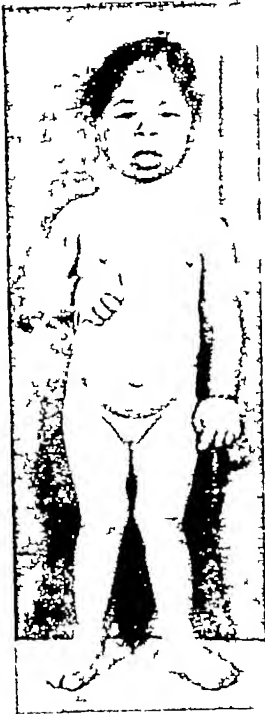


Fig. 4.5 Cretinoid dwarf

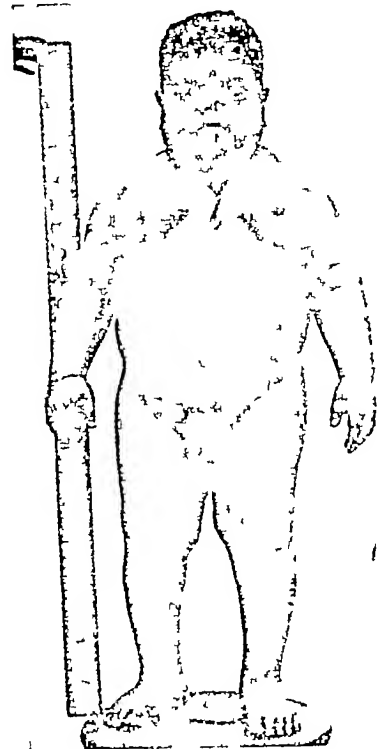


Fig 46 Dystrophia adiposo genitalis or Froehlich's syndrome

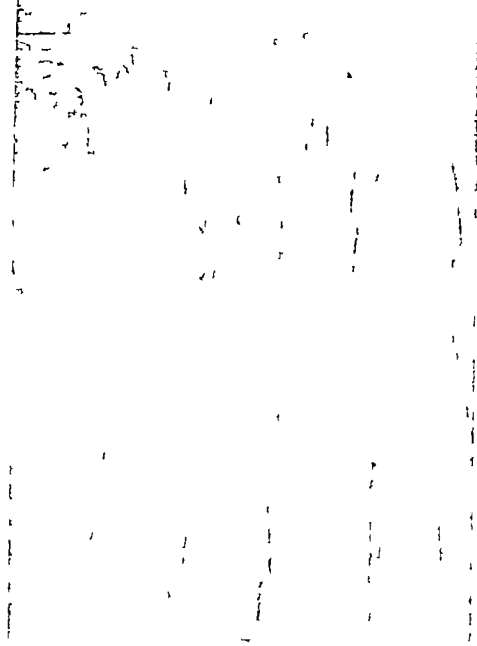


Fig 17 Pituitary dwarf with two  
normal individuals of same age



Fig 48 Gonadal  
dwarfism in adult  
female

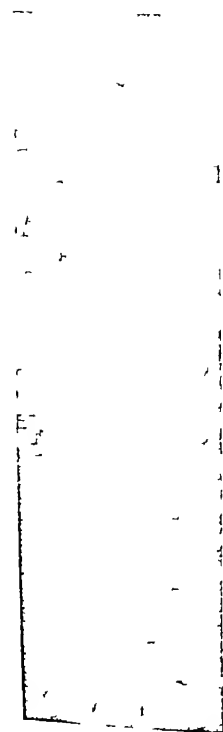


Fig 49 Precocious sexual development  
in adolescence—Macrogenitosomia praecox

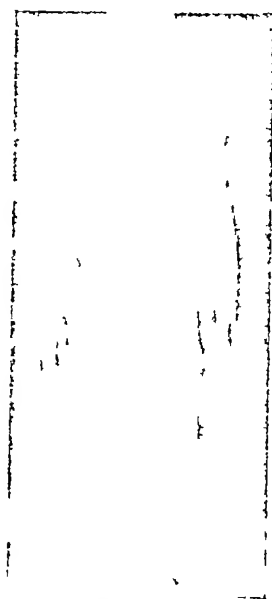


Fig 410 Achondro  
plastic dwarf

*Achondroplasia* The achondroplastic dwarf (Fig 4 10) is a common and easily recognizable variety of dwarf, with short and bowed legs and arms, a waddling gait, a small face with a flat nose, small, stubby and "trident" hands, normal intelligence and normal or excessive sex development. The dwarfism is due to a defective growth of cartilages.

*Osteogenesis imperfecta* (Fragilitas ossium, brittle bones) This condition is characterized by a short stature with markedly deformed and excessively fragile bones, a tendency to multiple fractures and deep blue sclerotics in a fair number of cases.

*Chondro-osteodystrophy* This condition, characterized by a short stature, an unusually short neck, pigeon breast and kyphotic deformity of the spine, is secondary to deformation with fragmentation of the epiphyses of the long bones and spine.

*Dyschondroplasia* There is a shortening of the long bones, secondary to "bowing" and other deformities.

*Gargoylism* (Lipocondro-dystrophy) The gargoyle is a short, mentally retarded individual with an angular kyphosis, an oxycephalic or hydrocephalic skull, a grotesque face, corneal opacities and enlarged liver and spleen.

*Other bone dysplasias*, which may cause dwarfism, are osteitis deformans or Paget's disease of bones, osteitis fibrosa cystica, hyperparathyroidism or von Recklinghausen's disease, renal rickets, renal osteodystrophy or osteofibrosis, osteopetrosis, marble or ivory bone disease or Albers-Schonberg disease, true rickets, Pott's disease of spine and acute anterior poliomyelitis.

**Cachetic dwarfism.** This may be secondary to either systemic, metabolic or infectious disease.

*Systemic dwarfism* Under this category are included (1) *Cardiac dwarfism*, usually secondary to congenital or rheumatic heart disease, the cardiac dwarf has a short stature and small genitalia but normal intelligence. (2) *Renal dwarfism* in children with chronic interstitial nephritis, the dwarfism is associated with decalcified and deformed bones, high blood pressure, polyuria, sallow complexion and ultimate renal failure. (3) *Cerebral dwarfism*, usually associated with microcephaly or hydrocephaly and idiocy. (4) *Coeliac dwarfism* (Fig 4 11) in cases of coeliac disease, associated with large, pale, fatty stools, abdominal distension and general wasting.

*Metabolic dwarfism* This may be secondary to (1) von Gierke's disease or glycogen-storage disease, the dwarfism being associated with infantilism and a large abdomen with a massive liver enlargement, or to (2) diabetes in childhood, overtreated with insulin, the short stature, large liver, sugar in the urine and history of hypoglycaemic attacks furnish the diagnostic clues.

*Infective dwarfism* This may be due to congenital syphilis, tuberculosis, malaria, hookworm infection, chronic ulcerative colitis or chronic respiratory disease. In congenital syphilis, the short stature is usually associated with the characteristic saddle-nose, Hutchinsonian teeth, rhagades at the angles of the mouth and interstitial keratitis.

## OTHER FORMS OF DWARFISM

**Achondroplasia** The achondro dwarf is a perfect miniature or 'pocket-book edition' of a well-proportioned normal individual, with normal sexual development and intelligence.

**Progeria (Hastings-Gilford disease)** A characteristic form of dwarfism with a wizened or senile appearance, small head and trunk, retarded sexual development, early graying or falling out of hair, disproportionate long extremities, thin and wrinkled skin, mental retardation, lack of adipose tissue, premature arteriosclerosis and early death.

**Ovarian agenesis (Wilton's disease)** The short stature is associated with webbing of neck, infantile sex organs, cubitus valgus and vascular anomalies such as coarctation of the aorta and high blood pressure.

**Lawrence-Moon-Biedl syndrome.** The dwarfism is associated with obesity, defective sexual development, mental retardation, polydactyly and retinitis pigmentosa.

## STATE OF NUTRITION

The state of nutrition depends mainly on the distribution of adipose tissue or fat in the body. On this basis individuals can be classed as *normal*, *overweight* (fat or obese) and *underweight* (thin or lean).

Factors regulating body weight have not been clearly elucidated. Obesity and underweight are problems of metabolism dependent on caloric intake, energy expenditure, metabolism of fat, protein, carbohydrate, minerals and water, appetite, mode of life, endocrine factors, heredity, constitution, personal habits, upbringing and environmental factors.

## OBESITY

The term obesity is applied to excessive deposition or distribution of fat in the body. Excess of fat is a disadvantage rather than an asset, it may "lengthen the waist line" but "shortens the life line" of the individual by imposing an extra burden on the cardiovascular system. Exogenous or alimentary obesity, from over-eating, is far more common than glandular or endogenous obesity.

Obesity may be mild, moderate or severe, or as wittily described by one author, "enviable", "regal" or "pitiable".

**Distribution of fat.** The following types of obesity are recognizable: (1) generalized type, (2) central or trunk type, involving only the trunk and neck, (3) superior or buffalo type, involving the face, neck, arms and upper part of trunk, (4) inferior type, involving the lower part of the trunk and legs (called lipodystrophia when accompanied by wasting of the upper half of the body), (5) girdle type, involving the hips, buttocks, abdomen and with a "fatty apron", (6) breeches or trochanteric type, involving only the buttocks as in the Hortelotor negro, (7) lipomatous type or multiple lipomata, with localized deposits of fat over the body, (called Dercum's disease or adiposa dolorosa when associated with tenderness and pain over the fatty lumps).

The type of distribution of fat may be of some diagnostic value. While generalized obesity is usual in alimentary or exogenous obesity, the superior or central type is common in Cushing's syndrome and hyperthyroidism, the girdle type in pituitary or hypohalamic obesity, and the trochanteric type in hypogonadal obesity.

## TYPES OF OBESITY

(1) **Exogenous obesity** A common form of obesity due to over-eating or excessive intake of food. The distribution of fat is uniform, although somewhat excessive under the chin ("double chin") and over the abdomen.

(2) **Endogenous or glandular obesity** Glandular in origin, it may be associated with Frohlich's syndrome, Cushing's syndrome, adreno-cortical hyperfunction, hypothyroidism, hypogonadism or the Lawrence-Moon-Biedl syndrome.

*Frohlich's syndrome* (Dystrophia adiposo-genitalis) A disease of adolescence or childhood with symmetrical obesity of "girdle" type, particularly over the abdomen, thighs, suprapubic and mammary regions. The fingers are narrow and tapering, axillary and pubic hair almost absent, complexion smooth and silky, voice effeminate, muscles hypotonic, intelligence normal and sex organs infantile.

*Cushing's syndrome* (Pituitary basophilism) A characteristic "buffalo" or central type of obesity, particularly of the face, neck and trunk with a sparing of the extremities, associated with hypertension, flushed face, diabetes mellitus, hypertrichosis (with a male type of hair distribution in females), amenorrhoea, purplish lines or lineae atrophicae over the lower abdomen, and with a tendency to bruise easily, headaches, body pains, decalcification of bones and kyphosis. Secondary to basophil adenoma of the pituitary or tumour of the adrenal cortex or thymus, Cushing's syndrome may start at any age after puberty.

*Adrenogenital syndrome* A condition of virilism or masculinism in females, associated with hirsutism or hairiness of the body and face (often with beard and moustache), coarse skin, increased musculature, amenorrhoea, manly voice, central type of obesity, tapering hands and feet and at times acne vulgaris and skin pigmentation. Secondary to adrenal cortical tumour, it may start before or after puberty, in its fully developed form, it may be indistinguishable from Cushing's disease.

*Diabetes of bearded women* (Achard-Thiers syndrome) A syndrome of obesity, diabetes mellitus and virilism (with characteristic hirsutism of face) in women, who appear "fat and bearded".

*Hypogonadal obesity* A peculiar type of obesity, mainly confined to the mons pubis, buttocks and thighs, may be noted in eunuchs (castrated males) or eunuchoids (with congenital hypoplasia or acquired disease of the gonads). It is associated with a smooth and delicate (alabaster) or coarse and wrinkly (senile) skin, hypotonic and poor musculature, hyperextensibility of joints, increased stature with long extremities, long and tapering fingers, infantile sex organs, female type of hair distribution and a high-pitched voice.

*Menopausal obesity* Hypogonadal obesity is common in women during or after the climacteric and is commonly associated with a mild degree of virilism, hirsutism, nervous and vasomotor instability, emotionalism and hypertension. The fat is deposited mainly over the neck, trunk and arms.

*Hypothyroid or myxoedematous obesity* In myxoedema, obesity is usually mild and confined mainly to the supraclavicular fossae, suprapubic region, wrists and ankles. The diagnosis is suggested by the dry coarse skin, malar flush, thick lips, tongue and eyelids, loss of hair over the head and eyebrows, spade-like hands, slow movements, slow pulse, apathetic dull expression and husky voice.

*Hypothalamic obesity* Obesity, arising in association with encephalitis lethargica, typhoid, tabes dorsalis and hydrocephalus, has been regarded as hypothalamic in origin.

Other forms of obesity include certain ill-defined forms of obesity of obscure aetiology such as

*Dercum's disease* (*Adiposis dolorosa*) Obesity associated with symmetrical, tender and painful fatty lumps over the body and a melancholic disposition

*Physiological obesity* A mild form of obesity observed temporarily during puberty or pregnancy

*Water salt obesity* Obesity secondary to retention of water or salt and characterized by sudden increases of body weight, which responds promptly to recumbency or diuretic therapy

*Lawrence Moon Biedl syndrome* A rare heredo-familial disease characterized by obesity, retinitis pigmentosa, syn- or polydactyly, mental deficiency and deformities of skull

*Hand Schuller Christian syndrome* A rare disease of lipid metabolism of childhood associated with obesity, dwarfism, gonadal hypoplasia, exophthalmos, and diabetes insipidus

*Localized obesity* In diffuse lipomatosis, fat is distributed as painless lumps all over the body, sometimes attaining gigantic proportions. In cervical lipomatosis, described in beer drinkers and alcoholics, large deposits of fat are more or less confined to the neck region ("double" or "triple" chin). In steatopygia, a lipomatous condition confined to certain African tribes, the obesity is confined to the buttocks

*Hyperinsulinism obesity* Obesity observed in cases of pancreatic tumour associated with attacks of spontaneous hypoglycaemia or in diabetic children overtreated with insulin

*Idiopathic or essential obesity* An obesity is labelled idiopathic or essential after all possible causes of gain in weight have been investigated and ruled out

## UNDERWEIGHT

Underweight, or a bodyweight well below normal average, may be due to (1) leanness, thinness or underdevelopment of fatty tissue, (2) emaciation, wasting or excessive loss of body fat, or (3) cachexia or emaciation associated with anaemia

## POSTURE

The position or attitude constantly assumed by a patient at rest or in motion is referred to as posture. The posture at rest or *static posture* may refer to the *sitting-up posture*, and the posture in bed or *decubitus*, or the upright, erect or standing posture

*Dynamic posture* or the posture of the body in action or motion includes such diverse activities as walking (or the gait), running, climbing, swimming and dancing

## STANDING POSTURE

The maintenance of a normal "standing" or upright posture is dependent on the integrity and coordination of the following neuromuscular mechanisms (1) adequate motor power in the muscles of the trunk and lower extremities, (2) postural sensory impulses regarding position of body, (3) labyrinthine impulses regarding spatial position of body and effects of gravity, (4) central

cerebellar coordinating mechanism, probably within the vermis, and (5) co-operation of higher centers (within the cerebral cortex) in the voluntary maintenance of posture. Loss of integrity of any of these mechanisms may result in defective standing or walking.

The attitude of a patient standing may be characteristic enough to suggest a diagnosis. Viewed from the side, the human body in the standing posture displays either a vertical line (good posture) or an S-shaped curve (poor posture). Alternatively, the normal standing posture may be classified as excellent, fair, poor or bad, it depends on the muscle tone and on the axial skeleton.

**Asthenic posture.** In asthenia, debility, wasting and senility, the normal curves of the spine are exaggerated resulting in a sunken chest, protuberant abdomen and drooping shoulders.

**Parkinsonian attitude.** In paralysis agitans or Parkinsonism, there is a characteristic attitude of flexion, involving the trunk and limbs, associated with immobility and poor associated movements.

**Hemiplegic posture.** There is a characteristic asymmetry, the affected side displaying adduction of the shoulder joint, flexion of the elbow, pronation and flexion of the wrist and hyperextension of the lower limb.

**Tabetic posture.** In tabes dorsalis, pseudoptosis, wrinkled forehead and small and unequal pupils, with wide separation of the feet may suggest the diagnosis.

**Lordotic posture.** In muscular weakness of neuromuscular origin, as in pseudo-hypertrophic muscular dystrophy (Fig 4 13), the back, when viewed from the side, may display a severe degree of lordosis.

**Flexibilitas cerea.** In schizophrenia, the body or limbs tend to maintain the same position for hours on end.

**Astasia abasia.** In hysteria, the patient may at times be unable to stand whilst able to walk or move the legs in bed.

Hysterical inability to stand differs from that due to organic nervous disease by (a) the absence of demonstrable organic involvement of the nervous system, (b) variability of defect from time to time, (c) aggravation of symptoms in the presence of spectators, (d) tendency to dramatize or exaggerate during standing tests.

**Cerebellar attitude.** In unilateral cerebellar disease (and in torticollis), the head may be held awkwardly or obliquely in relation to the body. Whilst mild cerebellar lesions may permit standing with difficulty, with the feet widely placed, severe lesions tend to incapacitate the patient from standing, because of wide swaying movements of the trunk.

**Immobile attitude.** A transitory immobility of the body may be noted in angina pectoris, peritonitis and flexibilitas cerea, persistent immobility suggests Parkinsonism or arthritis of spine. In ankylosing spondylitis (poker spine) the back is kept rigid and if the patient is asked to pick up an object from



the floor, he does so by bending his knees, the spine being kept absolutely rigid

### SITTING-UP POSTURE

This may be a posture of choice or the only posture possible in some cases of pericardial effusion, mediastinal new growth and cardiac failure

A sitting-up posture may not be possible with hip-joint disease and ankylosis, and in cases of severe hypotension or prostration

### DECUBITUS

The posture in bed or decubitus is often of great diagnostic and prognostic value as was first recognized by Hippocrates

**Normal decubitus** Normally, a patient in bed prefers to lie flat on his back, in the so-called "dorsal recumbent", or "normal supine" posture. This tends to be modified or altered in disease conditions

**Passive dorsal decubitus** During high or long continued fever, in states of debility or prostration (*status typhosus*), and in cases of frontal lobe tumour, the patient tends to constantly slip down in bed and with no effort at correction of the posture

**Rigid dorsal decubitus** The patient may lie on his back, in an immobile fashion, with flexion of one or both hip joints, in case of peritonitis, acute appendicitis, polyarthritis or hip-joint disease. Immobility of one or more limbs may simulate immobility of part or whole of the body

**Opisthotonus (Arched dorsal decubitus)** (Fig 4 14) The back is characteristically arched backwards, the weight of the body resting on the head and feet, which are buried deep in the pillows or mattress. This attitude is suggestive of tetanus and meningitis but may be encountered rarely in hysteria, strychnine poisoning or uraemia

**Emprosthotonus** The back is arched forward, instead of backward as in opisthotonus, the weight of the body resting on the forehead and toes. This may occur in tetanus, cerebrospinal meningitis, strychnine poisoning or Parkinsonism

**Pleurosthotonus** A lateral arching of the back associated with rigidity, may be observed rarely in cases of tetanus

**Lateral decubitus** This may be "right lateral" or "left lateral", according to whether the patient lies constantly on his right or left side. This type of decubitus may be due to *respiratory* disease (such as dry pleurisy, pleurisy with effusion, empyema, spontaneous pneumothorax, lung cavity or abscess, unilateral bronchiectasis or pleuropneumonia), *cardiovascular* disease with massive enlargement of the heart or liver, *neurological* disease (such as meningitis (Fig 4 15), head injury, cerebral irritation or sciatica), or *abdominal* disease, (such as appendicitis or appendicular abscess)

In *dry pleurisy*, the patient may lie either on the affected side in order to restrict the painful rubbing together of the inflamed surfaces of the pleura,

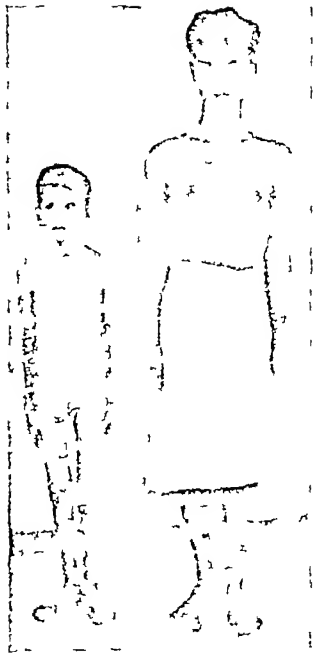


Fig 411 Coeliac dwarf with normal of same age

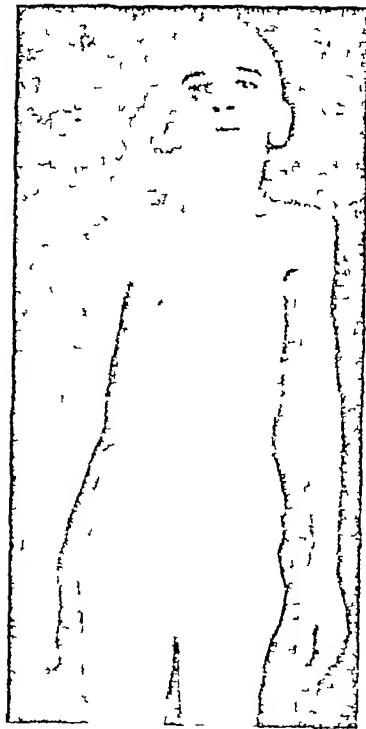


Fig 412 Case of progeria

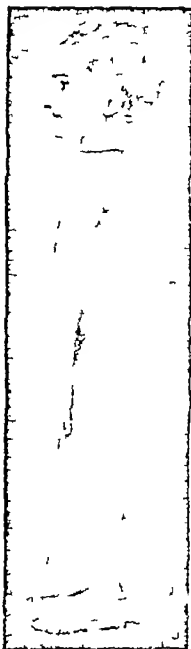


Fig 413 Lordotic posture in pseudo-hypertrophic muscular dystrophy



Fig 414 Opisthotonus in a child with meningitis

at de ub tus with photophobia in meningitis

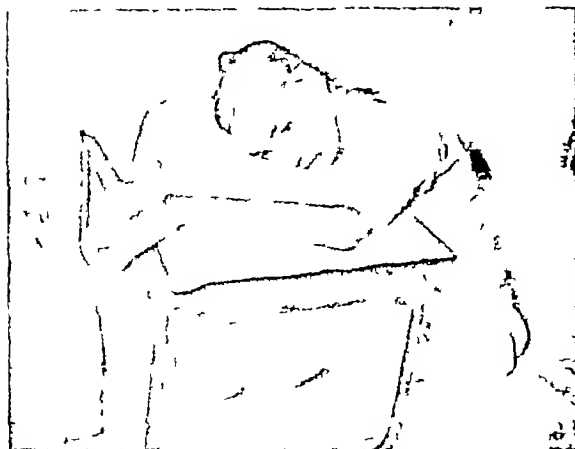


Fig. 416 Orthopnoeic posture in congestive cardiac failure

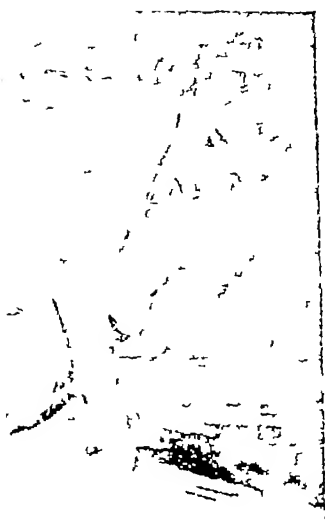


Fig. 417 Squatting position in Fallot's tetralogy

or on the good side in order to separate the two layers of the pleura, whichever posture proves less painful. In *pleural effusion*, pneumothorax and pleuropneumonia, the patient usually lies on the side of affection thus facilitating a proper expansion of the healthy lung. In *lung cavitation*, lung abscess communicating with a bronchus and in unilateral bronchiectasis, the patient automatically selects a position which allows the cavity to fill up rather than empty its contents into a neighbouring bronchus, thus provoking a bout of severe cough. In *heart disease*, the lateral decubitus is assumed in case of a massively enlarged heart or a painful and enlarged liver in order to prevent either postural dyspnoea or pain.

In amoebic abscess of the liver, the patient may lean or incline towards the left, in order to widen the right intercostal spaces with diminution of tension of the liver capsule.

**Coiled-up decubitus.** A lateral decubitus with the body curled up and the trunk and legs approximated, may be assumed in case of meningitis, cerebellar disease, cerebral irritation or abdominal colic.

**Orthopnoeic posture (Fig 4 16)** The patient, when dyspnoeic in the recumbent position prefers to sit up or be propped up in bed with pillows. With severe orthopnoea, he may insist on sitting up in a chair, night and day, with the arms resting on a table or on the edge of the bed and the head resting within his arms.

Orthopnoea may be encountered in cardiovascular diseases (such as cardiac asthma or left ventricular failure and pericardial effusion), respiratory diseases (such as pneumonia and bronchial asthma), intra-abdominal conditions (such as large ascites or a massive tumour), or in renal diseases (such as nephrosis, acute nephritis and uraemia).

**Prayers posture (Kneeling posture)** With extreme orthopnoea, as in some cases of massive pericardial effusion, severe congestive failure, aortic aneurysm or mediastinal tumour, the patient may lean forward either sitting in bed and usually embracing a pillow (pillow sign, position of Mohammedan prayer) or sitting on the edge of the bed with the legs hanging or resting on a chair. Sometimes the patient will remain standing with the trunk leaning forward, the arms on the back of a chair or the head of the bed. This has the objective of reducing venous return to the heart by the action of gravity and kinking the large veins at the groin.

**Squatting posture.** In Fallot's tetralogy, when the child gets out of breath or feels tired he sits on his heels with the knees pressed against the chest (knee-chest position or squatting on the haunches) (Fig 4 17). The child spontaneously adopts this posture and a very cyanotic child may squat most of the time. The plausible explanation for this is that in the crouching position, the abdominal aorta and femoral arteries are compressed or kinked, resulting in increased systemic arterial resistance and rise of aortic arterial pressure. The

latter, by offering greater resistance to the right-to-left shunt, allows more blood to flow, for oxygenation, from the right ventricle into the pulmonary circulation. Pressure on the inferior vena cava and trapping the venous return to the legs and reducing the amount of acid metabolites reaching the brain may also be a factor in relieving the dyspnoea.

**Prone position** Occasionally, a patient may prefer to lie in bed in a prone position or "on the stomach" as in cases of tuberculosis of the spine, acute gastroenteritis and through force of habit.

**Decubital restlessness** Extreme restlessness or tossing about in bed may be observed in cases of acute myocardial infarction, renal, intestinal or biliary colic, mania or delirium, drug-poisoning, Parkinson's disease, encephalitis, hyperpyrexia, chorea or thyrotoxicosis.

**Decubitus of thromboangitis** A characteristic sitting-up position with the legs hanging down and the head resting on the knees, even during sleep, is almost diagnostic of Buerger's disease or thromboangitis obliterans, this position affords partial relief from the agonizing pain in the lower extremities.

## THE SKIN

### METHODS OF EXAMINATION AND DIAGNOSIS

There are two circumstances which hinder the rational approach to diagnosis of skin diseases for a beginner, viz. (1) the attraction for a spot diagnosis based on the visual memory of clinical pictures of some diseases, e.g. psoriasis or lichen planus, and (2) the confusing similarity of eruptions which apparently look alike in many diseases of different genesis. The student should adopt a standard method of examination and gain practical experience to interpret the correct morphology of lesions. Lesions are the alphabets of cutaneous diagnosis and one can read them after sufficient clinical experience of seeing many patients. The books can only indicate a working method.

The examination of a skin case should be carried out as follows (1) history, (2) general examination and distribution of the eruptions, (3) the nature of lesions and their evolution, (4) relevant systemic findings, and (5) laboratory tests as and when indicated.

**History** The important points in a dermatologic history which should always be inquired into are as follows (1) The occupation of the patient particularly if there are eczematous eruptions on the hands and face (2) The family history of similar eruptions will indicate contagious diseases like scabies or sometimes leprosy, ringworm infections and pyoderma. It may also indicate familial and hereditary disorders (3) Previous history of similar skin disorders may indicate recurrent and chronic diseases such as metabolic disorders and allergy (4) History of having applied any medicaments which aggravated the eruption. This will give a clue to the diagnosis of drug sensitization which is liable to occur after application of sulphur, penicillin or strong irritants (5) History of having been given any drugs for any other condition

or of self medication (common self medications are aspirin, amidopyrine containing pain relievers, sulpha, phenolphthalein in chocolate laxatives) The drug eruptions cover a wide range of morphologic variations and should be considered as a "great imitator" of many cutaneous and systemic diseases Besides the above information, the usual details of a medical history like chronological enumeration of symptoms and other relevant data should also be ascertained

**General examination and distribution of eruptions** In addition to the usual details of a general examination carried out for a medical or a surgical case, the items of dermatologic interest are (1) the regional distribution of eruptions which will give an idea of the diagnostic clues afforded by the preponderant location of a dermatosis on certain areas, and (2) the involvement of the contiguous and accessory structures of the skin, viz hairs, nails, mucocutaneous junctions and the lymph glands, which will give an idea of the nature of a disease process, viz only involving the keratinized structures of skin, hairs and nails or spreading also to the deeper structures by lymphatics or arising systematically and involving the skin secondarily

**Local examination** This is the most important step in the recognition of a skin condition The *morphology* of the different varieties of skin lesions which one sees should be interpreted correctly because these eruptions constitute the signs on which are based the interpretation of symptoms The tables on pages 92-94 enumerate the common types of lesions and examples of diseases in which they are found The student should learn to interpret the morphology of lesions in different diseases by practical experience and get a correct mental picture of these For this, he has to learn the minute variations of similar lesions in different diseases also, for instance a papule of lichen planus is polygonal and violaceous in colour, that of syphilis is coppery brown and firm to feel, that of urticaria papulosa is indefinite and succulent and that of flat juvenile warts is rough, keratotic and superficial This then is the way by which the student should try to build up his dermatologic "vocabulary" and appreciate the variation of similar lesions in different diseases

The next important point to note about the eruptions is the *evolution of lesions* This gives an idea of the nature of a disease process For instance, a neoplastic lesion is likely to evolve to a steadily increasing size Destructive granulomatous lesions of chronic infections like syphilis and tuberculosis are likely to heal by scar formation, and a haemorrhagic macule will evolve with changes in colour To elicit the evolution the student should inspect the fresh lesions and compare them with the oldest lesions For such interpretation one only considers the primary lesions, i.e. those which arose primarily as a manifestation of a disease, and not the secondary lesions, i.e. those which arose by extraneous circumstances like secondary infection, scratching, exudation and some types of secondary pigmentations The secondary lesions then do not signify the eruptions which one associates with the genesis of a disease

TABLE I  
PRIMARY (ELEMENTARY) AND SPECIAL LESIONS

Types	Examples of conditions in which the type occurs
<b>FLAT LESIONS</b>	
1 The <i>Macule</i> is an abnormality of colour of skin that is not appreciably raised above the skin, i.e. a macule is flat and visible, but not palpable. The colour may be red, white, blue, brown, black, yellow, purple, or shades and combinations of colour (polychromatic). The size of a macule is variable (Figs 4 18, 4 19, 4 20, 4 21). Large areas of discoloration may require another descriptive term like a <i>patch</i> (of erythema, pigmentation, depigmentation, etc.)	Chloasma    Brown Flat pigmented naevi    Dark brown or black Freckles    Brown Vitiligo    White Syphilis    Coppery, palmar and plantar lesions of secondary syphilis von Recklinghausen's disease    Flat macular pigmentary naevi
2 <i>Haemorrhages</i> are extravasations of blood which may be further subclassified as petechiae (Fig 4 22), purpuras, ecchymoses, and haematomas	Trauma Some drug eruptions Many systemic diseases, e.g. blood dyscrasias, endocarditis, Schonlein-Henoch purpura, etc
<b>SOLID LESIONS</b>	
1 The <i>Papule</i> is a solid elevation on the skin that may be of normal or abnormal colour and is of the size of a pinhead up to a split pea (Fig 4 23)	Lichen planus    Violaceous, polygonal, shiny Moles and small neoplasms    Brown, flesh coloured and dome shaped Lichen chronicus simplex    Rhomboid papules Secondary syphilis    Coppery, yellowish brown papules Verruca juveniles    Flat rough papules
2. <i>Nodules</i> , <i>Tumours</i> and <i>Plaques</i> are also solid elevations on the skin but larger in size than papules. Nodules and tumours are more or less rounded, whereas plaques are flatter and may be of irregular geometric shape (Figs 4 24, 4 25, 4 26, 4 27, 4 28, 4 29, 4 30)	Erythema nodosum    Nodules Neoplasms    Nodules, tumours Drug eruptions    Nodules Syphilis    Tertiary lesions, e.g. gummas Leprosy    Nodules, plaques von Recklinghausen's disease    Nodules

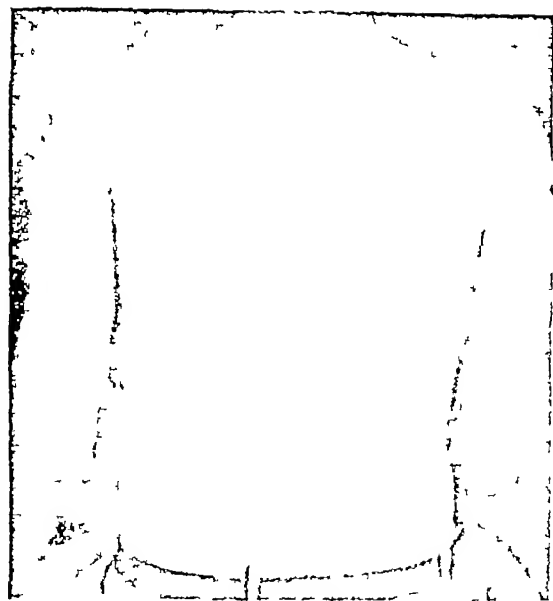


Fig 418 Pigmented macules of drug eruption



Fig 419 Brown small macules of naevus



Fig 421 Depigmented macules in vitiligo



Fig 420 Hypopigmented macules of tinea versicolor





Fig 4 23 Papules and nodules in lupus vulgaris



Fig 4 24 Deep seated nodules and ulceration in lupus vulgaris



Fig 4 26 Circinate lesions with hyperpigmented centre in secondary syphilis

Fig 4 25 Nodular and ulcerar lesions of secondary syphilis

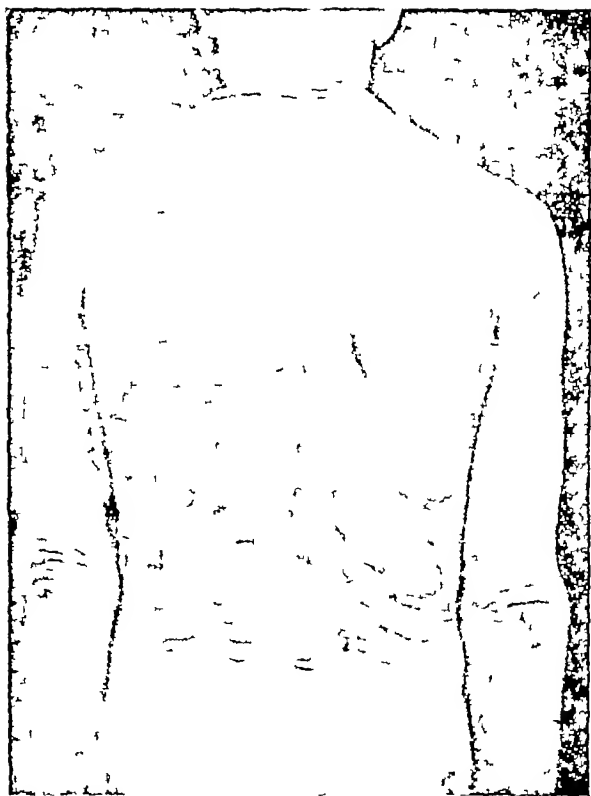


Fig 4 27 Circinate lesions of psoriasis



Fig 4 28 Multiple nodules and tumours in neurofibromatosis



Fig 4 29 Nodules of leprosy



Fig 4.30 Plaques of major tuberculoid leprosy

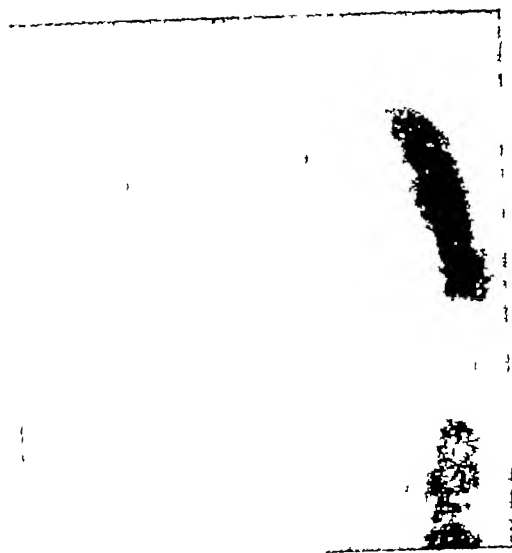


Fig. 4.32 Papillary lesions of wart

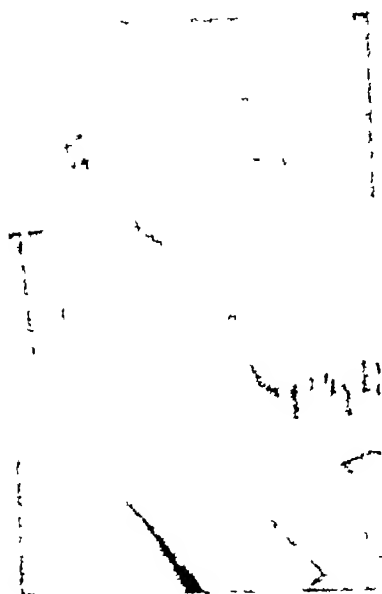


Fig. 4.33B Simultaneous herpes zoster and chicken pox eruption

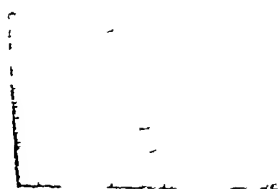


Fig. 4.33A Grouped vesicles of herpes

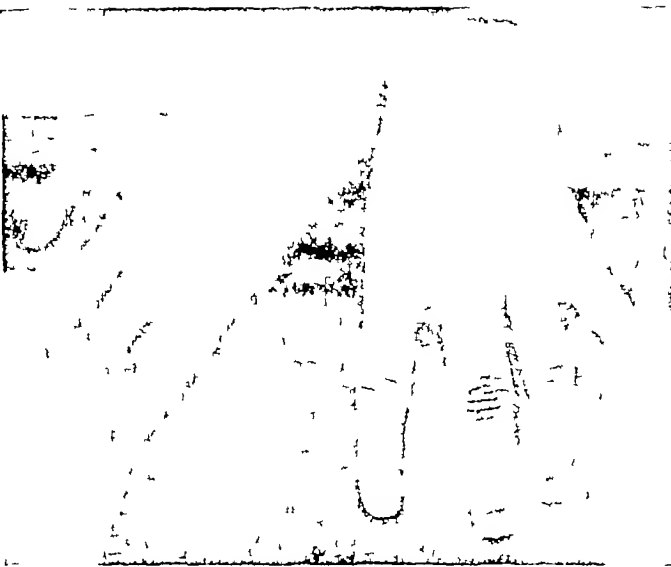


Fig 4.34 Vesicles and pustules in scabies

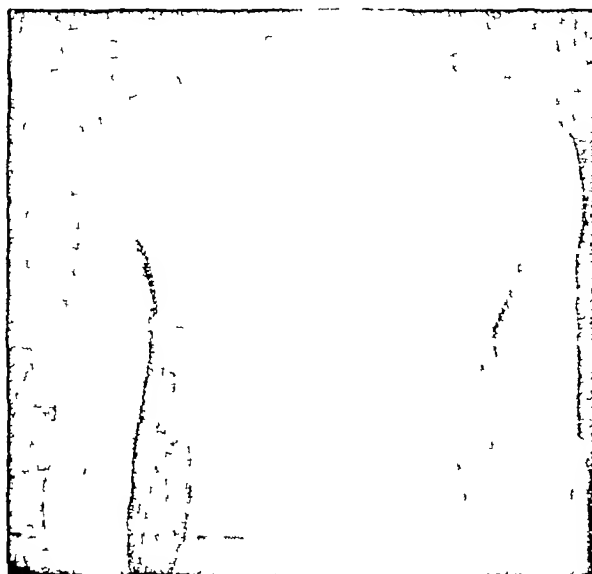


Fig 4.35 Vesicles and flat bullae in impetigo

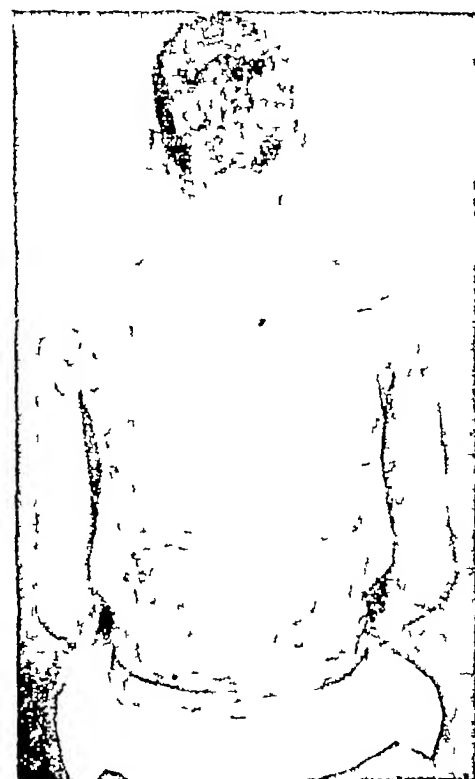


Fig 4.36 Broken bullae and raw areas in pemphigus



Fig. 4.37 Blebs in Stevens Johnson disease

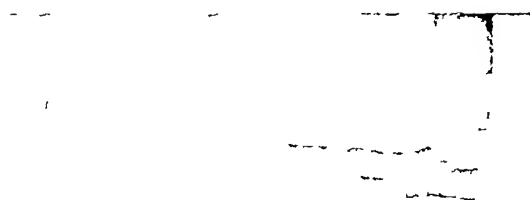


Fig. 4.38 Pus due to mollusc

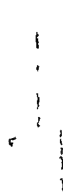


Fig. 4.39 Furuncle

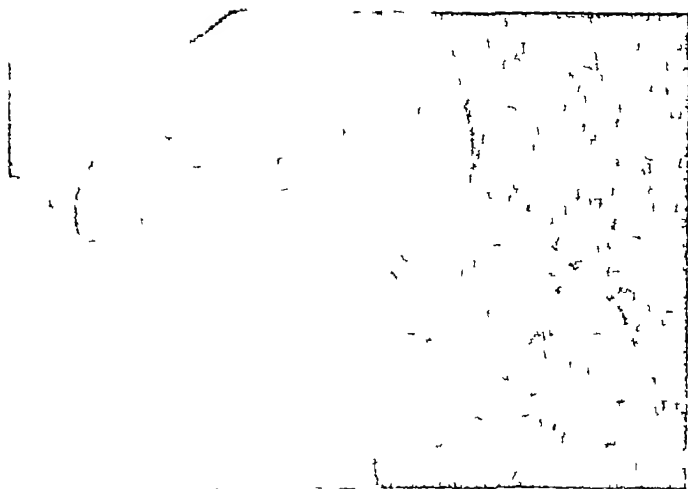


Fig 440 Vegetative lesions of bromoderma

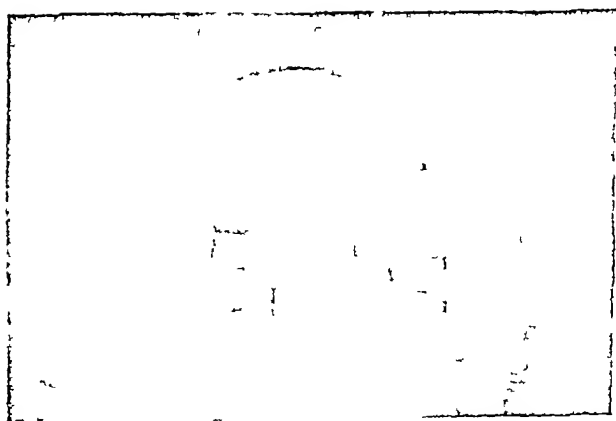


Fig 441 Dermographism



Fig 442 Erythematous and scaly patches of ringworm

Fig. 443 Scales in exfoliative dermatitis

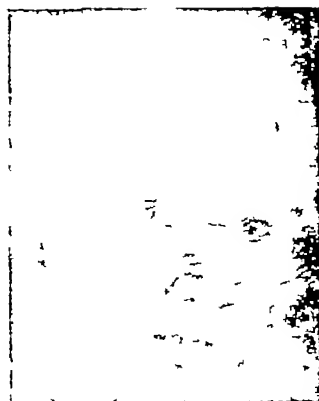
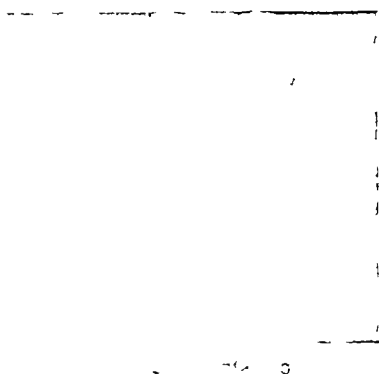


Fig. 445 Ulcerative lesion  
of basal cell carcinoma

Fig. 446 Xeroderma and  
ichthyosis of avitaminosis

TABLE I—(Contd)

Types	Examples of conditions in which the type occurs
3 The <i>Comedo</i> or <i>Blackhead</i> is a collection of sebum and keratin lodged in the pilosebaceous follicle with a black dot of oxidized fat visible at its top Generally in face Sometimes on extremities	Acne vulgaris Acneform dermatoses caused by tars and halides
4 The <i>Milium</i> or <i>Whitehead</i> is a papule of the size of a pinhead or millet seed which contains either sebaceous or cornified material It has no opening or communication with the skin surface	Colloid milia Sometimes around the healed bullae of pemphigus, epidermolysis bullosa and some other bullous diseases

## LESIONS WITH FLUID

- |   |  |
|---|--|
| 1 The <i>Vesicle</i> is an elevation on the skin that contains fluid (mostly serous) and is of the size of a pea or less (Figs 4 33A, 4 33B, 4 34, 4 35) Its position may be intra- or subepidermal                                   | <p>Ecze<sup>m</sup>a</p> <p>Herpes simplex and zoster</p> <p>Dermatitis herpetiformis</p> <p>Drug eruptions</p> <p>Chicken-pox and small-pox</p> <p>Pompholyx</p> <p>Some fungus infections</p>                                      |
| 2 The <i>Bulla</i> or <i>Bleb</i> has the same characteristic as the vesicle as to contents but is larger in size (Fig 4 36) <i>Blister</i> is a generic term for either a vesicle or bulla   | <p>Pemphigus vulgaris</p> <p>Erythema multiforme bullosum (Fig 4 37)</p> <p>Dermatitis herpetiformis</p> <p>Second-degree burns</p> <p>Drug eruptions</p> <p>Bullous impetigo</p> <p>Trophic bulla in leprosy and other neuritis</p> |
| 3 The <i>Pustule</i> is an elevation of the skin that has a purulent content and is of the size of a pea or less (Fig 4 38) Larger collections of pus are termed <i>Furuncles</i> (Fig 4 39), <i>Abscesses</i> , or <i>Carbuncles</i> | <p>Pyoderma (folliculitis, impetigo)</p> <p>Drug eruptions caused by iodides and bromides (Fig 4 40)</p> <p>Small-pox</p> <p>Acne vulgaris</p>   |

## EVANESCENT LESION

Only one lesion is of this nature The *Wheal* or *Urticaria* is a solid elevation of the skin that may be as small as a match head or as large as a palm Wheals are palpable, oedematous, frequently surrounded by a zone of erythema (flare), and are relatively evanescent (in the course of minutes to hours) After complete involution, the wheals leave no trace

Urticaria Acute, chronic, cholinergic, pigmented  
Insect bites and stings  
Angioneurotic oedema  
Dermographia (Fig 4 41)



TABLE II  
SECONDARY LESIONS

Secondary lesions evolve or develop from adventitious events like scratching, irritation and secondary infection of primary lesions, or as a result of subsidence of primary lesion

Types	Examples of conditions in which the type occurs
1 <i>Oozing</i> (weeping) results from exudation of serum in diseases with vesiculation due to rupture of these, or in other processes that damage the stratum corneum	Eczema Most vesicular and bullous dermatoses Abrasions Pyodermas Burns
2 <i>Crusting and Scabbing</i> result from the coagulation of serum and blood on the surface of the skin	Same as above and in severely pruritic dermatoses
3 <i>Excoriation</i> describes superficial or deep excavations of the skin caused by scratching or picking out the skin or the lesions	In all pruritic dermatoses, neurotic excoriations
4 <i>Scaling</i> (a) Visible exfoliation of the epidermis (Figs 4 42, 4 43) (b) Occasionally "invisible" but demonstrable on scraping	(a) In the involution of many dermatoses <i>Scaling is the early expression of a damage to epidermis from within or without</i> (b) <i>Tinea versicolor or early pityriasis rosea</i>
5 <i>Ulceration</i> is due to death of tissue en masse, may be superficial or deep (Figs 4 44, 4 45)	Trauma, especially if continuous Tuberculosis, syphilis, and other granulomatous diseases
6 <i>Scarring or Cicatrization</i> describes the replacement of lost substance by fibrosis	Following deeper ulcerations
7 <i>Atrophy</i> is the thinning out of skin due to destructive process from within. There is no loss of the superficial epidermis, but sweat (sebaceous) glands and hairs may be destroyed from within	Late leprosy or syphilis In scleroderma and acrodermatitis chronica atrophicans after the disease is burnt out In many congenital or hereditary dysplastic processes In senile skin
8 <i>Lichenification</i> describes thickening of the skin with surface markings of the skin made prominent	Chronic pruritic conditions like eczema and allergy Neurodermatitis
9 <i>Pigmentation</i> may be a secondary lesion as in post inflammatory pigmentation	Frequently in pigmented races in association with inflammatory lesions
10 <i>The Burrow</i> is an excavation or tract in the epidermis that may be (a) short and small, (b) long, large serpentine and solid	(a) <i>Scabies</i> (b) <i>Guinea-worm</i> (c) <i>Filaria</i>

**Relevant systemic findings** When being confronted with a skin eruption, the student should first ask the question whether the particular eruption is caused by circumstances outside the skin (such as physical and chemical trauma, microbial invasion or chemical sensitization), and hence is due to external agents or is due to systemic or internal derangements such as eruptive fevers, drug intolerance and metabolic derangements of diseases like diabetes or jaundice. The general clue to the above distinction is that the latter eruptions are more likely to be symmetrical or of a generalized nature and acute in onset, while the former are more likely to be localized, of a chronic nature and evolve slowly by contiguity. In case the student suspects internal causation then a thorough systemic examination as in a medical case is called for. This is also necessary for most chronic skin diseases of external causation to find out the involvement of deeper structures.

A few simple instruments are of value in the gross examination of the skin. These are (1) a magnifying glass to aid in the examination of small lesions, (2) a transparent microscopic slide to view the lesion after pressure, this helps in differentiating a pigmentary lesion from erythema and telangiectasis, (3) a mastoid curette to scrape the surface of a lesion for noting the details of the type of scales or for examination of these under microscope for demonstrating fungi, (4) a blunt probe for testing the consistency and tenderness of a lesion as well as to note the character of sinuses, (5) cotton and pin to test the sensations in a lesion, and (6) a syringe and needle to aspirate the contents of a lesion for diagnostic purpose.

**Laboratory methods** In addition to the use of laboratory procedures of routine nature, sometimes most extensive investigative approaches may have to be resorted to for a given case. The nature of investigation and the necessity for the use of such procedures has to be arrived at on the merits of an individual case.

Skin being an easily accessible organ, the causes of most infective disorders can be easily confirmed by examination of scrapings or smears stained appropriately. This is particularly for agents belonging to the classes of insects, protozoa, cocci, bacilli, fungi, spirochaetes and evidences of viral invasion. The details of such procedures should be looked for in the standard references in dermatology and pathology. In other conditions, a biopsy and histopathological examination of the disease by a competent dermal—histologist may be necessary.

## MOISTURE (SWEATING) OF SKIN

**Excessive dryness (Anhidrosis)** Abnormal dryness of the skin from loss of sweating may be found in dehydration (from cholera, severe diarrhoea, vomiting or diabetic coma), hypothyroidism, avitaminosis A (Fig 4.46), scurvy, scleroderma, xeroderma, ichthyosis ("fish skin"), old age, high fever or anasarca.

**Excessive moisture (Hyperhidrosis)** Excessive sweating is common during defervescence in fevers (especially malaria, septic fever and pneumonia), vasovagal attacks, acute myocardial infarction, rheumatic fever, hyperthyroidism, neurocirculatory asthenia, pulmonary tuberculosis (nocturnal or "night sweats"), Hodgkin's disease, diabetes mellitus, nervous excitement and during spells of hot weather or excessive humidity.

*Localized sweating* over the face (unilaterally) may be noted in trigeminal neuralgia, histamine headache, neurological disease or gustatory hyperhidrosis, and over the palms and soles in neurocirculatory asthenia nervousness, Raynaud's disease, general debility or hyperthyroidism

*Abnormal forms of sweating* (Dyshidrosis) Chromhidrosis or coloured sweat, usually yellow, may be noted in severe jaundice or hysteria. A urinous or ammoniacal sweat may be noted in uraemia, and haematidrosis or bloody sweat during anticoagulant treatment or haemorrhagic disease.

A *cold and clammy skin* (sweating with coldness) which is common in peripheral circulation failure, coronary thrombosis acute pain from any cause and in surgical emergencies such as generalized peritonitis, is frequently associated with a poor prognosis

## STATE OF THE SKIN

Wrinkled or loose skin often hanging in folds, may be found in old age (senility), progeria (premature senility) excessive loss of weight or fat, loss of oedema or eunuchoidism (hypogonadism) In *dehydration* the skin loses its elasticity and this may be demonstrated by the persistence of a pinched-up fold of skin or by the fine wrinkling which occurs when a broad fold of skin is compressed between finger and thumb

In *Pseudoxanthoma elasticum*, a rare inherited disease, the skin, particularly that covering the anterior aspect of the neck, axillae, groins and periumbilical area is wrinkled and extremely loose, and can be stretched much more than normal skin. The skin is also thickened, grooved and irregularly streaked by yellowish nodules which coalesce into plaques. The affected skin has been likened to coarse-grained moroccan leather or that of a plucked chicken. The skin changes are important because they may give a clue to diagnosis of haematemesis in otherwise apparently healthy patients

Tense or tight skin is found in scleroderma and over areas of inflammation, oedema or cellulitis

Excessive elasticity (hyperelasticity) of the skin and scalp may occur as a rare congenital disorder called Ehler's Danlos syndrome (Fig. 4.47). It is due to a deficiency of collagen and is characterised by the ability of the skin to stretch, hyperextensibility of the joints (double-jointedness) and by a bleeding tendency which may affect many internal organs. The skin is also fragile and heals with cigarette paper-like scars

Thickening or hypertrophy of the skin occurs in acromegaly, cretinism, myxoedema, avitaminosis A and C, elephantiasis, xeroderma, ichthyosis, neurofibromatosis and leprosy. In leprosy, the skin over the face is thickened and superabundant with a characteristic leonine appearance. Callosities on the hands in manual workers are examples of localized hypertrophy

Cutaneous changes in vitamin A deficiency consist of two distinct types of eruptions. 1. A goosepimple like eruption of dry, horny, round or oval



Fig 447 Hyperelasticity of scalp and skin (Ehlers-Danlos syndrome)



Fig 448 Toadskin or phrynodema from fatty acid deficiency



Fig. 449 Striae in Cushing's syndrome



sharply defined papules (Toad skin, phrynoderma, shark skin) occurring frequently on thighs and arms (Fig 4 48) Though the condition is usually attributed to vitamin A deficiency, deficiency of fatty acids may have something to do with it 2 Papular eruptions of pilosebaceous follicles This acneform lesion of vitamin A deficiency usually occurs on back and arms and less commonly on face and other parts of the body

Thinness or atrophy of the skin is common in old age (senile atrophy), circulatory or trophic disturbances, over venous oedema, in malnutrition and wasting diseases and as a primary disorder (idiopathic atrophy) The skin appears thin, shiny and glossy in appearance, white, grey or bluish in colour, and is inelastic and wrinkled like tissue paper

*Striae atrophicæ* or *lineæ albicantes* are grey or pinkish-white lines or scars of the skin, resulting from atrophy of elastic tissue after undue stretching They are common over the abdomen after pregnancy and in ascites and abdominal tumours, over the breasts after pregnancy and lactation, and over the thighs and buttocks after weight reduction Purplish striae over the lower abdomen are a characteristic feature of Cushing's syndrome

The striae, which may be sparse or abundant, are usually located over the outer aspects of the usually protuberant abdomen (Fig 4 49) At times they radiate peripherally from the axillary regions and laterally and medially from the areolæ of the breasts In contrast to the pearl-gray stretch marks of the obese or pregnant women, the purple striae of pituitary origin are typically depressed giving rise to a broad, furrowed effect

**Haemorrhagic phenomena** In patients who bruise or bleed easily, the basic defects may be (1) in the vessels due to *capillary fragility* as in scurvy, or allergic (Henoch Schonlein purpura) or senile purpura (2) *Deficiency of platelets* (thrombocytopenic purpura) with bleeding from nose, intestines, uterus, kidney, brain and into the skin This may occur as a primary event (idiopathic thrombocytopenia) or be secondary to bone marrow depression from drugs or marrow replacement by leukemia, reticulosis or cancer (3) *Coagulation defects* e.g. haemophilia, liver disease, or anticoagulant therapy Bleeding is often severe and can occur into muscles, from kidney or into the gut It may occur into the joints in haemophilia Haemorrhagic lesions of the skin are called *petechiae* if less than 2 mm in diameter, *purpuric spots* if 2 to 5 mm in diameter, *ecchymoses* if over 5 mm in diameter, *haematomata* if raised above the surface, *puncta* if minute in size, and *vibices* if linear in shape Haemorrhagic skin lesions, unlike those of erythema, cannot be obliterated by digital pressure or stretching, are pink or red in colour unlike areas of skin pigmentation and tend to change in colour and disappear unlike capillary naevi and skin pigmentations

**Tattooed skin** Diseases that localise in tattoos include secondary and tertiary syphilis, lichen planus, psoriasis, lupus erythematosus, and sarcoidosis

**Subcutaneous oedema** Excessive accumulation of fluid in the subcutaneous tissues is called oedema. Oedema may be either *localized* (to small areas, one or more extremities or face) or *generalized* (affecting the whole body). Generalized oedema is often called *anasarca* or dropsy. In the common type of so-called *venous oedema*, the overlying skin is smooth, shiny, cold and bluish, the oedema pits on pressure and the regional lymphnodes are unaffected. In *lymphatic oedema* (from lymphatic obstruction, as in filariasis), on the other hand, the overlying skin is rough or pitted and warm, there is no pitting on pressure, and regional lymphnodes are frequently involved.

A *pitting* oedema can be demonstrated by exerting sustained pressure with the thumb over the oedematous area, on withdrawal of the thumb, a "pit" or "depression" is observed for some time at the site of pressure.

**Dermatographia** (Autographism, dermatographism, factitious urticaria) (Fig 4 41) In subjects of urticaria or allergy, firm stroking of the skin with a pointed object or finger-nail results in a red linear elevation followed, within a few minutes, by a wheal surrounded by a diffuse pink flare or flush.

**Subcutaneous emphysema**, due to the presence of gas or air in the subcutaneous tissues, gives a characteristic *crepitation* or crackling on palpation of the affected region. This may be in the region of the thorax, abdomen, neck or extremity and may be due to rib fractures, penetrating wounds or infection by gas-forming organisms.

**Black dermatographia** This term is applied to the black lines or streaks sometimes observed on the skin, after the wearing of cheap trinkets or jewellery or after being stroked with a metallic object. The condition is attributed to a mechanical rubbing off of the metal by a dry, non-oily skin.

## COLOUR OF SKIN

The normal colour of skin depends mainly on the amount (both absolute and relative) of five pigments, viz melanin, melanoid, oxyhaemoglobin, reduced haemoglobin and carotene and, to some extent, on the optical phenomenon of "scattering". Primitive man being of tropical or subtropical origin is supposed to have had a brown skin, brown eyes and black or dark brown hair. The blonde skin and blue eyes of the Nordic races of today are due to gradual loss of skin pigment through centuries of climatic influence.

**Decrease of lack of pigmentation** Depigmentation or lack of normal skin pigment may be due to albinism or vitiligo.

**Albinism** A congenital disease of unknown aetiology, characterized by complete and usually generalized absence of pigment from the skin and other epidermal structures. Albinos usually display a white or pinkish-white skin, pink irises, white or colourless hair, photophobia and nystagmus. *Naevis achromicus* is a rare form of localized or segmental albinism.

**Piebaldism** This is a patchy variety of albinism and is present at birth. The lack of pigmentation may be confined to small areas of the scalp.

**Vitiligo (Leucoderma)** (Fig 4 21) An acquired and frequently progressive condition of unknown aetiology characterized by large or small dead-white patches on the skin with sharp margins and surrounded by areas of hyperpigmentation. The patches are particularly common over the face, hands and thighs, and tend to enlarge and multiply. The white patches of leucoderma display neither anaesthesia (as in leprosy patches), nor induration (as in scleroderma), nor variability of colour (as in pinta). Vitiligo occurs not infrequently in Addison's disease where depigmentation and pigmentation is interchanged in irregular patches. It is also not uncommon in hyperthyroidism, diabetes mellitus, pernicious anaemia, and adrenocortical insufficiency.

**Increased pigmentation** Increase in pigmentation of the skin may be *generalized* or *localized* and due to *endogenous pigments* (viz melanin, haemoglobin, haemoglobin derivatives and lipochrome) or *exogenous pigments* (e.g. metallic, chemical or biochemical substances).

### Diffuse or generalised pigmentation

(1) **Yellow pigmentation** (a) Jaundice is the most common cause of yellow skin colour, the colour being due to excess of bilirubin. (b) Carotenæmia due to excess of carotene is best seen in the palms and soles. It is absent on the sclera and bulbar conjunctiva. (c) Drugs such as tetracycline, mepacrine. (d) Chronic uraemia. (e) Yellow chemicals used in industry can stain the exposed parts of the skin, such persons are described as 'industrial canaries'. (f) Picric acid ingestion, or its absorption from skin ointments. (g) Diffuse xanthomatosis. (h) Lesions of urticaria pigmentosa may give a yellow colour.

(2) **Haemoglobin pigmentation** In darker racial groups, the palms and soles, finger nails, lips and palpebral conjunctiva should be inspected for colour change due to haemoglobin because of minimal or lack of melanization in these areas.

(a) Cyanosis (See p 102)

(b) Polycythaemia Flushed slightly cyanotic appearance

(c) Carboxyhaemoglobinaemia Peculiar cherry-red colour due to exposure to CO<sub>2</sub>

(d) Methaemoglobinaemia Chocolate blue skin colour

(e) Sulph-haemoglobinaemia Mauve-like colour

(3) **Melanin pigmentation (Melanosis)** The degree of melanization normal for a person of one racial group may be abnormal for another.

(a) External causes Exposure to sun (tanning), ultraviolet rays, photosensitization of skin to actinic rays by application of certain substances such



as dihydroxyacetone, exposure to roentgen rays or radium. Also mechanical irritation of the skin e.g. severe pruritus as in Hodgkin's disease or application of hot water bottles to abdomen.

(b) Internal causes (i) Nutritional Pellagra, often scurvy. In vitamin A deficiency pigmentation is confined to the hyperkeratotic follicular lesions (ii) Of hormonal origin Pituitary and adrenal 'Acromegaly, Addison's disease, after adrenalectomy, ACTH in large doses Gonadal Oral administration of oestrogens Thyroid Hyperthyroidism. (iii) Neurological lesions: Neurofibromatosis, congenital neurocutaneous syndrome of melanosis of skin and CNS (iv) Skin diseases: Any skin disorder associated with an inflammatory response may be followed by melanosis (v) Miscellaneous A generalised increase in pigment may also be due to scleroderma, dermatomyositis, and chlorpromazine and chloroquine treatment.

(4) *Metallic pigmentation* Argynia due to deposition of silver. The colour is accentuated by sunlight. Deposition of gold (chrysiasis) and bismuth (bismuthia) are rare and simulate argynia.

*Localized or patchy pigmentation* This may be due to (1) melanin deposition, as in sunburn, freckles, varicella's disease (chase infection), leucoderma, von Recklinghausen's disease or neurofibromatosis ("café-au-lait" patches), tuberous sclerosis and pheochromocytoma, acanthosis nigricans, callositas, after burns, friction or scratching, pigmented naevi and melanotic sarcoma (2) haemoglobin and its derivatives, as in ecchymoses, bruises, purpuric spots (3) Epithelioma as in xanthelasma or xanthoma (4) exogenous pigments, as in tattooing marks, bismuth, mercury or lead poisoning (6 use line on gums especially) and perfume dermatitis (from the local use of Eau de Cologne)

*Special types of pigmentation.* In Addison's disease, the pigmentation is usually dark brown or black due to the deposition of melanin pigment in the basal cells of epidermis, is of gradual onset, variable in intensity and shows a special predilection for areas exposed to light (e.g. hands, face and neck), friction areas and pressure points (e.g. under belts, garters, trusses and especially in the girdle region), normal pigmented areas (e.g. genitals, nipples and areolae), warm areas of folds (e.g. groins, axillae and under the breasts), creases of the palms and mucous membranes of the gums, cheeks, palate and vagina. Buccal pigmentation of the Addisonian type (melanoplakia) may also be due to 2 Negro ancestry.

In *pellagra*, due to prolonged dietary deficiency of the P.P. factor, the skin lesions (Fig. 4.50) are dark brown or yellowish brown, symmetrical, most marked on the exposed parts, elbows, knees, neck and perineal regions, sharply demarcated from normal skin areas and associated with glossitis, stomatitis and mental changes. Skin lesions may be entirely lacking in the so-called *pellagra sine pellagra*.

*Ir vitamin B<sub>12</sub> deficiency* one type of pigmentation has come to be recognized as specific. It is deep brown or brownish-black, most pronounced on the

hands and feet, associated with varying pigmentation of other parts of the body-generalized pigmentation, irregular pigmentation of face and pigmentation of buccal mucous membranes, and patchy pigmentation of soles and palms, and fine spotty pigmentation of medial aspects of thighs and arms

*Kwashiorkor* is a disease of infants and young children due primarily to deficient quality and quantity of protein in food. It is characterized by oedema, skin changes and retarded growth. The skin changes consist of pigmentation, drying, desquamation and sometimes weeping areas. The dry crackled skin particularly on the forearms and legs has been likened to crazy pavement (Fig 4 51)

In *haemochromatosis* (*bronzed diabetes*), the skin pigmentation is bronze, bluish-grey or slate coloured, due to deposition of haemosiderin and haemofuscin in the Malpighian layer and sweat glands of the skin, is maximal over exposed parts, skin folds, genitalia and over the hands and forearms and associated with cirrhosis of the liver and diabetes mellitus. Melanin and copper are also said to be deposited in this disease.

In *chloasma*, a condition frequently associated with pregnancy, there are patches of increased pigmentation (so-called "liver spots"), particularly over the forehead (the "mask of pregnancy"). This has also been seen in females taking the contraceptive pill.

In *freckles* (*lentigo* or *ephelis*), particularly common in red-haired and white-skinned individuals exposed to the rays of the sun, there are small multiple spots of pigmentation on the exposed parts. When found over covered areas of skin, they are called "cold freckles", in old age, freckles may lead to senile keratosis and carcinoma of skin.

In *acanthosis nigricans*, a rare disease sometimes associated with advanced malignancy, there are characteristic black or brown, smooth and velvet-like patches of pigmentation.

In *generalized xanthomatosis* of skin, "chamois-leather-like" golden yellow areas of pigmentation, secondary to deposition of cholesterol and cholesterol esters, may appear over the skin surface, particularly over the elbows and eyes.

**Pallor.** Deficiency or lack of the normal colour of the skin or mucous membranes is called pallor. Contrary to lay belief, pallor is not always associated with anaemia. Pallor may be due to defective quality or number of red blood cells (anaemia), peripheral vasoconstriction, or accumulation of fluid (as in oedema) or myxomatous substance (as in myxoedema and cretinism) in the tissues.

Pallor of the skin may be *habitual* (as in racial or familial pallor), *progressive* (as in anaemia or bacterial endocarditis), *temporary* (as in purpura or after haemorrhage) or *evanescent* (as in nausea or syncope).

*Special types of pallor* have been recognized in certain diseases, e.g. an ivory white or pasty white skin in nephritis and nephrosis, a waxy skin in lardaceous disease, an alabaster or semitranslucent skin in certain acyanotic forms of congenital heart disease and in eunuchoidism, a muddy or earthy tinge of skin (sallowiness) in chronic malaria, malignancy and cachexia, a cafe-au-lait appearance in subacute bacterial endocarditis, a greenish pallor in chlorosis, and a lemon-yellow or biscuit colour of the skin in cases of pernicious anaemia and subacute combined degeneration of the spinal cord, a Dresden china complexion in aortic regurgitation.

The *clinical causes* of pallor of the skin and mucosa are many and varied. Pallor may be racial (as in the Chinese), familial, climatic (tropical), environmental (in urban populations), or occupational (as in radium and lead workers). It is frequently due to pathological conditions, like anaemias and other disorders of the blood (e.g. iron deficiency anaemia, pernicious anaemia, aplastic anaemia and leukaemias), haemorrhagic phenomena, infections like tuberculosis and bacterial endocarditis, tropical diseases like malaria and kala-azar, malignant diseases, chronic suppurations, renal diseases (e.g. nephritis, nephrosis or pyelonephritis), cardiovascular diseases like coronary disease or aortic valve disease, alimentary diseases like colitis or duodenal ulcer, and endocrine diseases like Simmond's disease and myxoedema.

**Redness of skin** Redness of the skin is usually due to hyperaemia or excessive content of blood in the superficial capillaries. *Dusky redness* is either due to an associated cyanosis (imperfect oxygenation of blood in the capillaries), polycythaemia (increased number of red blood cells) as in Osler-Vaquez disease, or spider naevi or vascular angiomata (as in cirrhosis of liver).

*Diffuse redness* of the skin may be evanescent or transitory (e.g. after a hot bath, massage, exercise, exposure to heat, cold, alcohol, drugs or emotion), temporary (e.g. during fevers, scarlatina, skin eruptions, wounds, burns and sun-burn) or persistent (e.g. polycythaemia vera).

*Localized redness* of the skin may be noted in skin eruptions, wounds, acute lobar pneumonia (one or both cheeks), Horton's syndrome of histamine headache (unilateral flushing of face), mitral stenosis ("mitral flush" on both cheeks), acute pulmonary tuberculosis ("hectic flush" on both cheeks), myxoedema ("malar flush" with surrounding pallor), Raynaud's phenomenon (over the hands) and cirrhosis of the liver (vascular angiomata on nose and cheeks).

*Unusual persistence or intermittent flushing* of the skin may result from carcinoid tumor of the intestine with hepatic metastasis.

**Cyanosis** Cyanosis is more often a physical sign than a symptom. The term cyanosis is usually applied to bluish or violet discoloration of the skin and mucous membranes, due to absolute increase (over 5 g per 100 ml) of reduced haemoglobin in the blood of the sub-capillary venous plexuses of the skin. Cyanosis can be best observed in the feet and hands where the skin is thin and less pigmented, and where the capillaries are numerous—cheeks, ears, lips, nail beds and in the mucous membrane of the mouth.

In anaemia, if the haemoglobin is less than 30 per cent, cyanosis cannot occur because even if all the haemoglobin is in the reduced form within the capillaries, the total amount would still be less than the critical level of the 5 gm per cent.

*Mechanism of production* According to the mode of production, four main types of cyanosis can be differentiated, viz. (1) *anoxaemic* cyanosis, due to inadequate oxygenation

of blood in the lungs, from low oxygen tension in the alveoli (as in mountain sickness or obstructed airway), unhealthy alveolar walls with decreased permeability to oxygen (as in diseases of the lung parenchyma) or from inadequate or shallow breathing (as in alkalaemic conditions), (2) *stagnation* cyanosis, due to increased dissociation of oxyhaemoglobin, from a sluggish circulation (as in Raynaud's disease, thrombophlebitis and extreme cold), (3) *shunt* or *admixture* cyanosis, from a venous-arterial shunt (as in the cyanotic forms of congenital heart disease), and (4) *replacement* cyanosis due to replacement of the oxygen-ion of oxyhaemoglobin (as in meth- and sulph-haemoglobinaemia) This is not a true cyanosis

## CLINICAL TYPES OF CYANOSIS

*Central cyanosis* This results from increased arterial oxygen unsaturation either from cardiac or pulmonary lesion or both Central cyanosis is detected in warm sites such as the conjunctivae or inside the mouth When it is chronic and severe it is associated with polycythaemia and clubbing

*Peripheral cyanosis* is due to increased utilization of oxygen by the tissues thus increasing the venous oxygen unsaturation It results from stagnation of blood and hence is seen in the extremities or ear lobes, although if the patient is cold, the lips too may be blue in absence of central cyanosis It does not lead to finger clubbing or polycythaemia.

*Mixed cyanosis* A case of cyanosis may present in some degree of both central and peripheral cyanosis For instance, in cor-pulmonale due to chronic emphysema or fibrosis of lung, the lung lesion tends to produce central cyanosis whereas the associated right-sided failure tends to cause a peripheral type of cyanosis

*Differential cyanosis* The term differential cyanosis is applied to the condition where some part of the body receives more hypoxic blood than others Cyanosis confined to the lower extremities with little or no cyanosis of the arms and face, is practically diagnostic of pulmonary hypertension with right-to-left shunt through a patent ductus arteriosus Cyanosis confined to the upper extremities with little or no involvement of the legs is suggestive of transposition of the great vessels, with shunting of oxygenated blood from the pulmonary artery (through a patent ductus arteriosus) into the aorta, but if the ductus is proximal to the left subclavian artery the right hand is less cyanosed than the left hand and the feet

*Local cyanosis* Cyanosis confined to an extremity or part of an extremity (particularly the hand or forearm) should suggest the possibility of Raynaud's syndrome, scleroderma, peripheral artery obstruction, thrombophlebitis, phlebothrombosis or varicosities of veins

In cyanosis, the *colour* of the skin and mucosae is not invariably blue, it may be blue-black or black (as in cases of Ayerza's disease or "cardiacos negros", crush injuries of the thorax or abdomen, or congenital heart disease with massive venous-arterial shunt), heliotrope as in the once dreaded condition of influenzal pneumonia, grey, ashen or "corpse-like" (as in peripheral circulatory failure, coronary thrombosis and phosgene poisoning), purplish

or "plum-coloured" (as in polycythaemia and Raynaud's disease), mauve or lavender (as in sulph haemoglobinaemia), chocolate or brownish blue (as in meth haemoglobinemia), cherry-red (as in carbon monoxide poisoning), bright-red (as in cyanide poisoning) or even green (as in cyanosis associated with icterus)

The *intensity* of cyanosis, in a given case, may vary from time to time, depending on circumstances. In some forms of congenital heart disease, the cyanosis may first make its appearance in the second decade of life or even later (*cyanose tardive*)

## CONGENITAL ABNORMALITIES

### Haemangiomas

(Naevus, or birth mark) A congenital and localized abnormality of the nature of hypertrophy or hyperplasia of some element of the skin or subcutaneous tissue. Both vascular and non-vascular naevi are recognized. A vascular naevus may be either a (1) *capillary naevus* ("port-wine stain") (Fig 4 52) a pinkish-red or purple patch of skin, flush with the surface and caused by dilated superficial capillaries or (2) *cavernous naevus* ("strawberry mark"), a raised, red or purple, soft and compressible lesion caused by massively dilated and

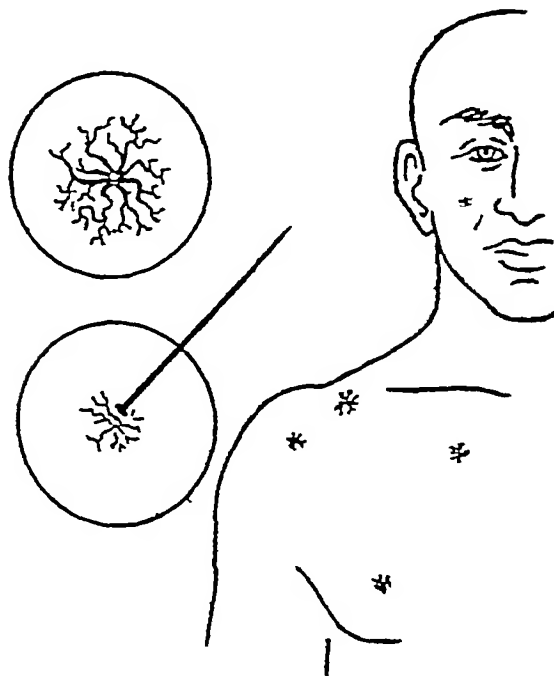


Fig. 4 54 Spider angioma. Pressure on the central prominence of the arterial spider with a pin causes blanching of the whole lesion

thickened capillaries), or (3) *arterial spider* (spider angioma, vascular spider, spider telangiectasis), so called because it is shaped like the body and legs of a spider with a central arteriole from which radiate hair-like branches for a distance of about 0.5 to 1 cm (Fig 4 53). For some unknown reason the common sites for the occurrence of arterial spiders are the neck, face, arms and upper trunk, in other words in the area of distribution of the superior vena cava, they are rarely seen over the lower trunk and legs. Pressure on the central prominence with a pin causes blanching of the whole lesion (Fig 4 54). Pulsation of the central core can be demonstrated by applying counter pressure over the area with a glass slide. These little vascular abnormalities are not true naevi because they usually occur in late life and it is not



Fig 4.50 Pigmentation of exposed areas in pellagra

Fig 4.51 Crazy pavement skin in Kwashiorkor. Note the oedema



Fig 4.52 Naevus

Fig. 4.53 Spider naevus

Fig. 4.54 See p. 104

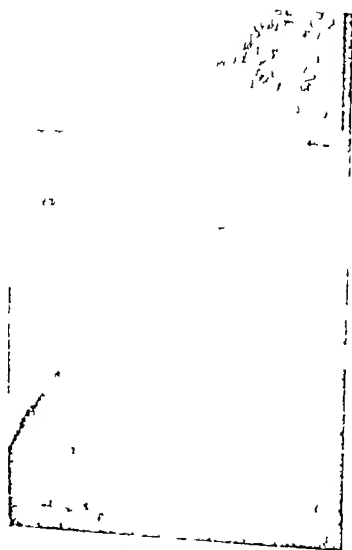


Fig. 4.55 Keloid



Fig. 4.56 Xanthelasma round the eyes

yet clear if they result from a congenital malformation. Normally the arterioles supplying the skin stop at the vascular plane deep in the dermis, a spider naevus results when an arteriole comes right up to the surface. In addition to arising spontaneously they also occur in cirrhosis of the liver. They may occur transiently in virus hepatitis. They may also be found in pregnancy, avitaminosis B, thyrotoxicosis, rheumatic fever, rheumatoid arthritis, xeroderma pigmentosa, chronic irradiation dermatitis, lupus erythematosus and Cushing's disease. This valuable sign of liver cirrhosis becomes more intense and increases in size and number during progression of the disease with a tendency to improve or subside during clinical remission.

Small vessels scattered in the skin are often associated with arterial spiders, their distribution being more or less the same. They have been compared to the silk thread in American paper money and the skin is therefore called *paper-money skin*.

A *non-vascular naevus* may be flat or raised, soft or hard, pigmented or non-pigmented, hairy, warty or smooth, and small or large enough to involve the upper or lower half of the body ("fur cape" or "bathing-drawer" type). Pigmented moles are particularly important as they may become malignant later in life.

**Moles** Moles are the most common congenital abnormality of the skin and vary in size from pin-head size papules to lesions covering a large portion of the skin. They may be flat or raised above the surface, pigmented or flesh coloured, hairy or non-hairy. There are several varieties. (a) *Epidermal naevi* commonly occur on the scalp. On other parts of the body, the raised flesh-coloured or pigmented papules may be arranged in a linear distribution. (b) *Pigmented mole* (benign melanoma). These are usually less than 0.5 cm in diameter, may be slightly raised and produce a cluster of coarse dark hairs.

**Ichthyosis:** This is characterised by dryness of the skin (Fig 4.46). In severe cases the skin resembles that of a crocodile. Types: (a) *Ichthyosis vulgaris* is the most common type, the lower limbs being most affected. There is some thickening of the skin of the palms and soles with exaggeration of the lines of the palms (so-called palmist's despair). (b) *Bullous ichthyosiform erythroderma*, a rare condition in which the picture varies from localised areas of hyperkeratosis of flexures and periumbilical region to gross involvement of the skin (porcupine man). (c) *Ichthyosiform erythroderma* (Collodion foetus). This may be present at birth in which case the baby is covered by a film of collodion.

**Infantile eczema** Usually arises between third and six months of life. The face is first affected and the rash soon extends to involve most of the body, the striking feature being the intensity of the irritation. The affected skin is reddened, scaling and excoriated and in severe cases covered with thick crusts.



## TUMOURS OF THE SKIN

## Benign tumours

**Verrucae (Warts)** A wart is a small circumscribed epidermal growth caused by a virus. The types are (a) *Verrucae vulgaris* the common warts seen on hands of children and young adults (b) *Verrucae filiformis* small, slender threadlike growths on neck and eyelids and about the mouth (c) *Verrucae digitata* a group of fingerlike projections with horny caps found often on face and neck (d) *Verrucae plana* (Juvenile warts), flat or dome-topped growths on face, forehead and backs of hands, wrists, and knees (e) *Verrucae accuminata* (venereal warts). An accumulation of filiform warts which form cauliflower masses and occur on genitals and in perianal regions.

**Plantar warts** Plantar warts are flat growths on sole of the foot. There may be painful solitary warts on metatarsal heads or the heel, large primary with many satellite warts or the mosaic wart.

**Seborrhaeic warts** These usually appear on scalp, fore head and trunk in middle-age and increase in number with age. They start as slightly raised brownish macules and subsequently become more raised, darker and have a warty surface.

**Sebaceous cyst (Wen)** A small or large retention cyst of a sebaceous gland with a central dimple or pit and particularly common over the scalp.

**Dermoid cyst (Inclusion dermoid)** A congenital and benign cystic tumour frequently containing hair or sebaceous glands, and commonly arising on the face at the sites of fusion of embryonal segments.

**Lipoma** A small or large, rounded, fatty tumour within the subcutaneous tissue, arising on any part of the body surface, is called a lipoma. In *multiple lipomatosis*, large number of lipomata are scattered all over the body. Multiple and painful lipomatous masses in conjunction with obesity are typical of Dercum's disease.

**Neurofibroma ("Fibromatous naevus")** Small or large, soft or hard, sessile or pedunculated connective tissue tumours, arising from the skin surface or from underneath the skin and associated with peripheral nerves, are called neurofibromata.

**Multiple neurofibromatosis (von Recklinghausen's disease)** A hereditary disease, due to congenitally defective ectoderm, and characterized by (1) *cutaneous pigmentation* (small or large areas of brown or cafe-au-lait pigmentation of the trunk), (2) *cutaneous fibromas* (soft, pink, small or large, sessile or pedunculated skin tumours particularly over the trunk and face), (3) *peripheral neurofibromas* (small or large, bead-like, movable, and frequently tender nodules, palpable along the course of superficial cutaneous nerves, particularly of the limbs), (4) *pachydermatocele* or *elephantiasis neuromatosa* (cutaneous or subcutaneous tissue hypertrophy resulting in large masses

hanging down in folds, especially affecting the head and neck and associated with "plexiform" or "diffuse" neuromata), (5) *kyphoscoliosis*, enlarged skull and hyperostosis of facial bones

**Morgan's spots** (Cherry angioma) These spots are bright red in colour, flat or slightly elevated and occur commonly on the front of the chest and on the abdomen. Morgan's spots increase in size and number with age.

**Keloid** (Fig 4 55) A firm, smooth and shiny, white or discoloured ridge or plaque on the skin, either arising spontaneously or secondarily to some injury, burn, sinus or scar. It is caused by hypertrophy of the connective tissue of the corium. After attaining a certain size, the lesion tends to become stationary or retrogress.

**Xanthomas:** These are small tumours, yellow or orange in colour, often associated with hyperlipoproteinaemias, but some types, especially xanthelasmas may be found in people with normal lipid profiles. There are four different types: (1) *Xanthelasmas* Circumscribed yellowish plaques in or around the eyelids, (Fig 4 56), usually at the inner canthus, but may be diffuse and spread on to the upper border of the cheek. (2) *Tuberos xanthomas* Nodules often yellowish and coalescing mostly over knees and elbows. (3) *Tendon xanthomas* Often multiple and indurated, these occur over elbows and on tendo Achilles, knees and extensor surfaces of hands and feet. (4) *Plane xanthomas* Usually raised and well circumscribed they may be found on palms, neck and chest.

All the above forms of xanthoma may be associated with either hypercholesteremia or hypertriglyceridaemia. The following types are associated with hypertriglyceridaemias only. *Eruptive xanthomas* They may appear in crops commonly on buttocks, arms, and legs. They are raised with a yellowish centre and often an erythematous margin. *Tuberous xanthomas* These are intermediate between tuberous and eruptive types, may look inflamed and may coalesce. *Palmar xanthomas* These give the appearance of smeared peanut butter on palms and palmar surfaces of fingers.

**Molluscum contagiosum.** Small, multiple, shiny, pearly-white or pink, epithelial tumours of viral origin, arising anywhere on the body surface.

**Senile seborrhoeic adenomas.** These appear after middle age as small round yellowish white firm nodules on face especially forehead and cheeks.

**Pyogenic granulomata** (Granuloma telangiectaticum) are well defined raised nodules which bleed easily on minor trauma and commonly occur on the hand.

**Histiocytoma.** These small raised slightly pigmented nodules commonly occur on women's legs.

**Keranto-acanthomas** These occur on face, sometimes on body and hands of elderly individuals, each resembling a small marble surmounted by a keratinous plug.

**Milium (Miliaria)** All types of miliaria are caused by interference with the overflow of sweat on to the surface of the skin. Miliaria crystallina (sudamina) are seen in young babies and after sunburn as small clear superficial vesicles. Miliaria rubra (prickly heat) arises on skin normally covered in hot humid conditions. Miliaria profunda is a more pronounced form of prickly heat.

**Adenoma sebaceum (Fig 4 57)** A rash consisting of partly confluent papules over bridge of the nose and 'butterfly' area of the face and over the chin particularly near the lower lip often seen in epiloia (tuberous sclerosis). Adenoma sebaceum, epilepsy and mental retardation constitute the triad of this disease.

### Malignant tumours

**Epithelioma (Squamous-cell carcinoma)** The commonest variety of malignant tumour of the skin, it usually supervenes on some pre-existing skin lesion like a wart, an old radium or X-ray burn, solar dermatitis, chronic irritation from arsenic or tar, longstanding lupus vulgaris or senile keratosis. It is particularly common over the lips and ears, usually starts as a tiny pink or white nodule which later ulcerates. Its main characteristics are the slow growth, craggy hardness, indurated base, an adherent crust or serosanguinous discharge, a pearly-white indurated border, regional lymphnode enlargement, its solitary nature and appearance after the age of forty years. It usually originates in the prickle-cell layer of the skin, later invading the deeper layers.

**Rodent ulcer (Basal cell epithelioma) (Fig 4 45)** The lesion appears as a small, semitranslucent, pearly nodule, usually on the face. After growing for years, it usually develops into a typical "rodent ulcer" with a raised or "rolled" semitranslucent pearly edge and a shallow encrusted base. Occasionally, ulceration is replaced by a "cystic" or "cicatricial" type of lesion.

**Malignant melanoma (Naevus or melano-sarcoma or -carcinoma)** One of the most virulent of all malignancies, the malignant melanoma usually arises from an old pigmented and hitherto innocent mole anywhere on the body surface. Malignant change in a mole is suggested by a suddenly increasing induration, ulceration, crust formation, bleeding, regional lymphnode enlargement or by metastatic involvement of the brain, liver or skin. The transition from benign to malignant may be initiated by irritation, trauma or pregnancy. The outlook of malignant melanoma is extremely gloomy, death being usually a matter of months.

**Intra-epithelial epithelioma (Bowen's disease)** In this condition, the neoplastic epidermal cells spread outward instead of penetrating into the deeper tissues, the growth remaining confined to the epidermis. Its main characteristics are the slow and superficial development, plaque-like appearance, reddish-brown colour, and the smooth velvety and crusted or raw ulcerated



Fig 4.57 Adenoma sebaceum

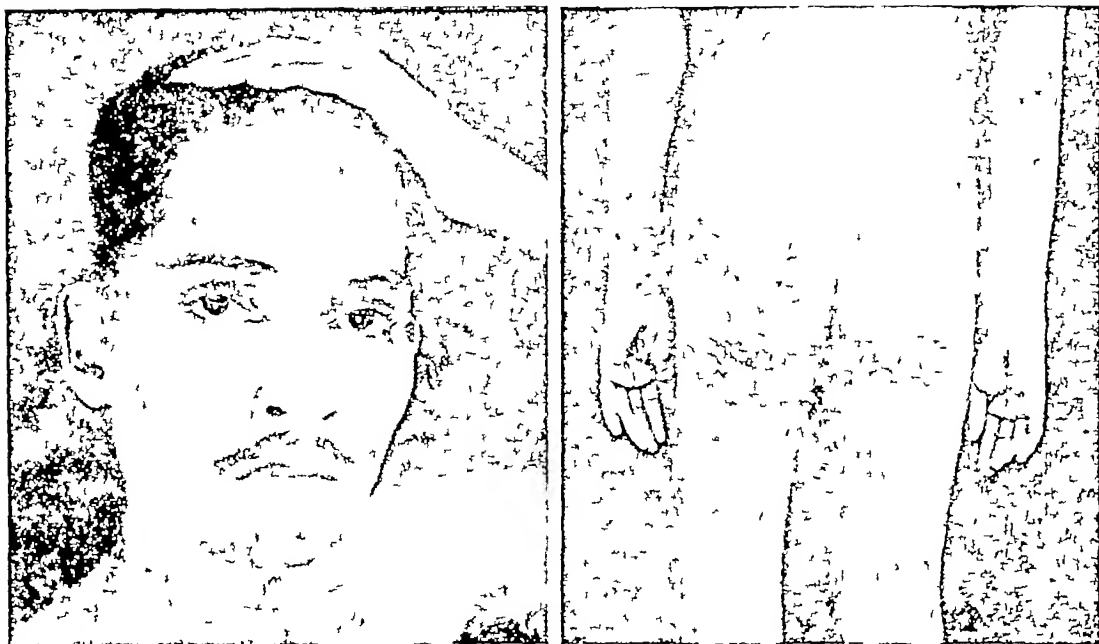


Fig 4.58 Leukaemids (leukaemia cutis)



girl of 11 years with adrenal neoplasm. Note the  
enlarged clitoris

surface It may affect the nipple or areola of the breast (*Paget's disease*), the glans penis or the skin or mucous membrane in any part of the body

**Mycosis fungoides** A rare and chronic disease which starts as an intractable pruritus, goes through a so-called "pre-mycotic stage" of skin eruptions (which may be eczematous, psoriasis-like, urticarial or erythrodermic), then develops into "tomato-like" red or purple skin tumours, and finally ulcerates and fungates (mycotic stage) with a fatal termination

**Metastatic skin tumours** These may appear in the form of single or multiple metastatic nodules in the skin or subcutaneous tissues, in cases of carcinoma, malignant melanoma, lymphoblastoma or leukaemia (leukaemids) (Fig 4 58) The condition usually proves rapidly fatal

#### DISTRIBUTION OF HAIR

Hair growth is influenced by genetic, racial, nutritional and hormonal factors There are three varieties of hair (a) *Non-sexual* (neutral) hair which is under the control of growth hormone and is found in both sexes on the scalp, eyelashes, forehead and lower parts of the body (b) *Ambisexual hair* This appears in both sexes at puberty under the influence of adrenal cortex and the gonads (c) *Male type hair*, which appears over the pubic triangle, beard region, ears, tip of nose, chest and trunk under the influence of testosterone The rate of hair growth and shedding is under the influence of thyroid hormones

#### Excess hair

**HIRSUTISM** means excess growth of hair of male type It occurs mainly in women as one of the manifestations of virilism, but is also seen in boys with precocious puberty

**HYPERTRICHOSIS** is overabundance of body hair but not of male type It may be (1) *Primary or idiopathic* form which is racial or familial (2) *Sexual precocity*—The appearance of hair growth may be premature (3) *Nonendocrine conditions*—Certain nonendocrine conditions may be associated with excessive hair growth—pulmonary tuberculosis, patients paralysed by poliomyelitis, spina bifida particularly with paralysis of lower extremities, epileptics with grand mal seizures, porphyria, hemochromatosis, physiological increase in hair-growth occurs during pregnancy (4) *Endocrine causes*—(a) *Hypothyroidism in childhood*—In the young hypothyroid child, one of the cardinal signs pointing to hypothyroidism is the presence of an increased amount of lanugo hair on the back with an increased amount of hair growth on the forearms (b) *Adrenal hyperplasia and neoplasm* (Figs 4 59, 8 19B) Adrenal neoplasm should be suspected if there is a history of rapid onset and progression of hirsutism (c) *Ovarian factors*—Ovarian hirsutism is characteristically concentrated in the area of the face rather than all over the body

(5) *Iatrogenic factors*—Administration to female patients of androgens, anabolic steroids, ACTH and corticosteroids, hydantoimates, streptomycin, penicillin and diazoxide (6) *Climacteric and postmenopausal hirsutism* (7) *As part of stress syndrome* particularly after excessive burns (8) *Morgagni's syndrome*—characterized by obesity in a climacteric patient with a Cushing like habitus and in addition hyperostosis frontalis interna plus an extensive degree of hirsutism. (9) *Haemochromatosis*

**Heterosexual distribution of body hair** A male type of distribution of pubic hair ("male pubic triangle") or the appearance of hair on the face and chin in female patients, although occasionally encountered in normal subjects, is usually associated with some glandular disorder (inter-sexuality)

The opposite condition of a female type of distribution of pubic hair (horizontal upper border in a male patient) is also suggestive of glandular disorder

A general reduction or absence of body hair may be due to hypogonadism, hypopituitarism (Fig 4 60), hypofunction of the adrenal cortex or familial

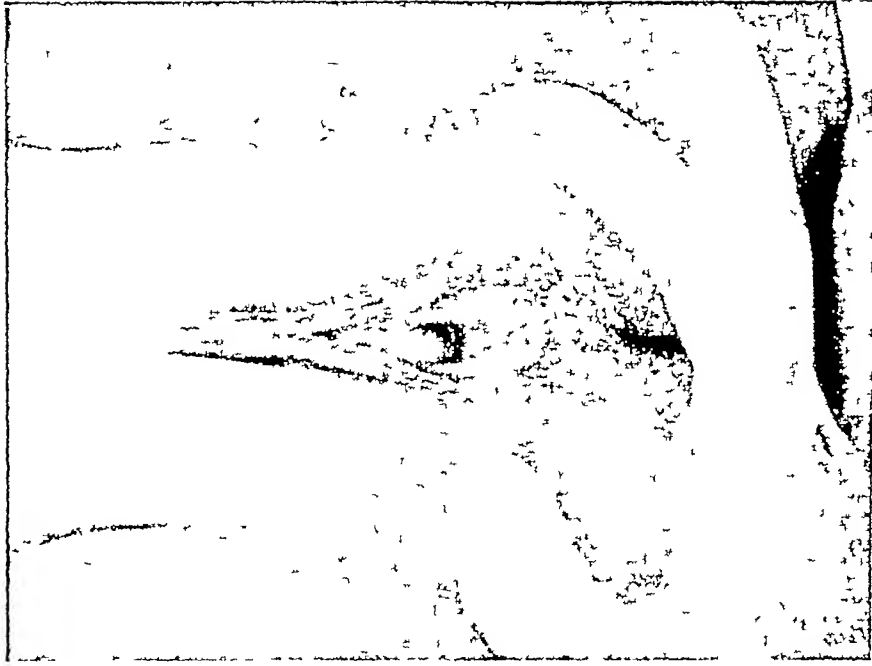
A general loss of body hair (*alopecia universalis*) may be glandular in origin (hypothyroidism, hypoparathyroidism or hypopituitarism), familial or of unexplained origin

## SKIN ODOUR (BODY ODOUR)

Olfactory diagnosis or recognition of disease by the body odour was an art practised and explored for years by ancient generations of physicians. Body odour may be *physiological*, as in pregnancy during menstruation, overweight or obesity, excessive sweating and unclean habits. *Racial* odours (dependent on dietetic habits and distribution of sweat glands) may be distinctive in some cases (e.g. the so-called blubber-like odour" of the Eskimos the "feculant odour" of certain Baltic races, the "buttery odour" of Americans and the "odourless" Chinese and Japanese). *Pathological* causes of distinctive body odours are many and varied. Amongst the most important may be mentioned certain infections e.g. acute rheumatic fever (acid" or sour smell), typhus fever (mousy smell), Vincent's stomatitis, diphtheria, follicular tonsillitis, small pox, measles and acute caseous pneumonia, poisons, e.g. cyanide, carbon monoxide, exhaust gas, lysol, arsenic, mercury, lead and gasoline, addictions, e.g. alcohol tobacco and opium certain occupations, e.g. workers in arsenic (garlic' smell) selenium, tellurium and workers at gas stations, and skin conditions, e.g. athlete's foot, fungus infections and gangrenous areas, bromhidrosis and urhidrosis

**Bromhidrosis** (or osmidrosis) A stinking and offensive sweat odour, particularly over the armpits and feet, is common in anaemia, debility and alcoholism.

**Urhidrosis** An ammoniacal or urinous smell of the skin sweat in cases of uraemia and rarely in normal subjects

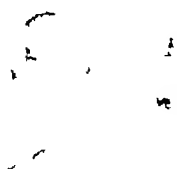


**Fig 460** Sheehan's disease Note myxoedematous appearance loss of axillary and pubic hair, atrophy of breasts and genitalia (Courtesy Dr P N Shah, Indian Cancer Research Centre, Bombay)





Fig. 52 Continued



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Fig 55. Otoliths of fish.

# 5 | The Head

## THE SKULL OR CRANIUM

### SIZE OF SKULL

The size of the skull is usually measured circumferentially along lines drawn horizontally backward from the supraorbital ridges to the occipital protuberance. The average circumference of the skull is 13 inches at birth, 18 inches at the age of 1 year, 20 inches at age of 7, 21 inches at age of 15 and 22 inches in an adult.

**Abnormalities of size.** The skull may be unduly small as in microcephaly (Fig 5 1) or generalized craniostenosis, (Fig 5 2) or unduly large as in hydrocephalus, osteitis deformans, acromegaly or gargoylism. In achondroplasia, the skull, although normal in size, appears unduly large in view of the short extremities.

**Microcephaly** may result from cerebral agenesis, toxoplasmosis, cytomegalic inclusion disease, etc.

**Macrocephaly** is less common than microcephaly. It may be due to megalencephaly or to hydrocephalus. The head may start to enlarge soon after birth. Mental defect is the rule.

**Hydrocephalus** (Fig 5 3). The head is unusually large and globular with the front and sides sloping outwards, eyes pushed downwards with the sclerotics exposed above the iris (sunset sign), large forehead, somewhat flattened vertex, and distended veins with a scant growth of hair over the scalp. In infancy and early childhood, hydrocephalus is associated with widely open, bulging and even pulsating fontanelles, separated sutures and prominent scalp veins. A hollow "cracked-pot" note may be elicited on percussion of the skull. The circumference of the skull may be 30 inches or more.

*Skull in osteitis deformans* (Paget's disease) (Fig 5 4) The skull becomes progressively larger in adult life, the patient requiring larger and larger sizes of hats. The head circumference becomes larger than normal, the forehead prominent and the head is held forward, the face appears unduly small, the back is arched and legs bowed and thickened, imparting an "ape-like" attitude to the patient. The skull has been likened in shape to an "acorn" in this disease.

*Acromegalic head* (Fig. 5 17) The head and face are larger than normal and the supraorbital ridges are prominent with a receding forehead. An "acromegalic facies" is associated with the large head and makes recognition easy.

*Achondroplasia* A disease of foetal life with defective endochondral ossification, resulting in a short stature or dwarfism, with relatively short extremities and a head that appears unduly large in contrast to the body.

#### SHAPE OF SKULL

*Craniosostenosis* A congenital abnormality with premature closure of the cranial sutures resulting in malformation of the head and in some cases to an increase of intracranial pressure (Fig 5 2). The shape of the skull will depend on which sutures are involved.

*Oxycephaly* (*Oxycephalus*, "tower skull", "steeple head" or "sugar-loaf head") (Fig 5 5) This is a characteristic type of vertically elongated head with a pointed vertex, a wide forehead, shallow orbits with bulging eyes, proptosis or exophthalmos and a vacant expression. The antero-posterior diameter of the skull is markedly reduced and the vertical diameter increased. This condition results from early closure of the coronal and sagittal sutures and is sometimes familial (Fig 5 6).

*Acrocephaly* The head is high and pointed but wide at the base. Pressure symptoms are less common than in oxycephaly. It may be associated with syndactyly of the hands and sometimes of the feet in Apert syndrome. It is also seen in cranio-facial dysostosis (with beaked nose, hypoplastic maxilla, short upper and protruding lower lips).

*Scaphocephaly* (Fig 5 7) The skull in this condition grows in length and height but not in breadth due to synostosis of the sagittal suture.

"Boxy head" ("Rachitic head") In rickets, the skull appears larger than normal and tends to be oblong or square with prominence and thickening of the frontal and parietal eminences ("bossed head") and slight flattening of the vertex (Fig 5 8).

*Basilar invagination* (*Platybasia*) This is a deformity of the skull in which the head appears to lie in extension on a shortened neck (Fig 5 9). It is due to upward displacement of the basilar and condylar portions of the occipital bone with consequent reduction in the size of the posterior fossa. The



Fig 56 Familial oxycephaly



Fig 57 Scaphocephaly (Dolicocephaly)

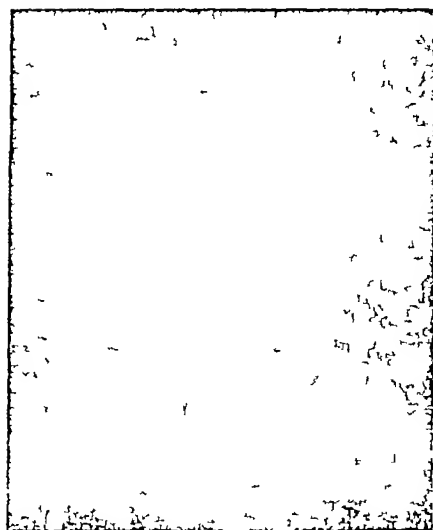


Fig 58 Boxy head of rickets

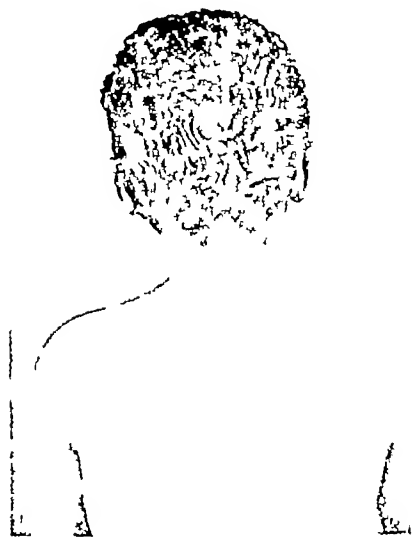


Fig 59 Platybasia Note the short neck and low hair-line

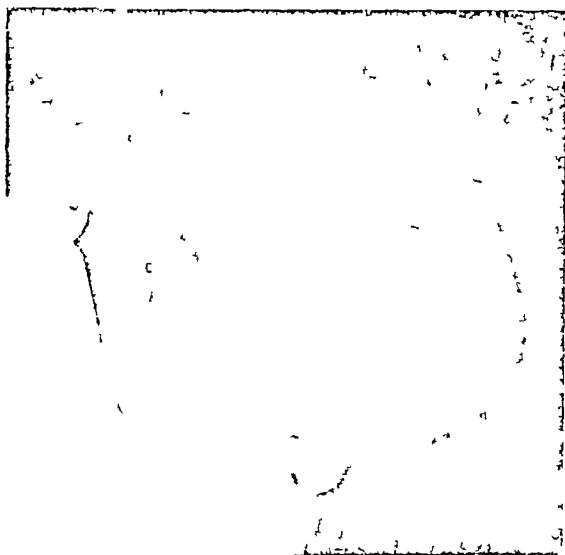


Fig 510 Skull in craniocleido-dysostosis

Fig. 511 Meningocele

Fig. 512 Plexiform neuroma of scalp

Fig. 513 Old aneurysm of scalp

Fig. 514 Metastatic deposits of skull with primary carcinoma of thyroid gland

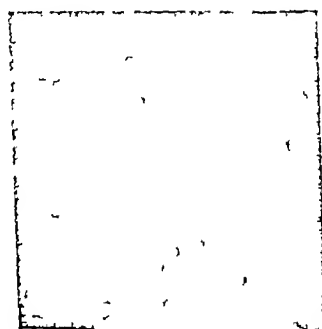


Fig. 515 Lipoid deposits at base of skull in Hand Schüller-Christian syndrome

deformity is usually congenital in origin and is often associated with other malformations such as fusion of two or more of the cervical vertebrae (Klippel-Feil syndrome) or stenosis of the foramen magnum. Basilar impression may also result from softening of the occipital bone in osteogenesis imperfecta, rickets, osteomalacia, Paget's disease and hypoparathyroidism.

**Cross-bun skull** In congenital syphilis, the head displays a vertical forehead, sunken bridge of nose, and prominence of the frontal eminences (*Parrot's nodes* or *frontal bosses*) giving a characteristic "cross-bun" appearance to the head. The forehead and face appear characteristically "flat" or squarish.

**Gargoylism (Hurler's syndrome)** In gargoyles, the skull is large (due to hydrocephalus), abnormally shaped (usually scaphocephalic), the bridge of the nose depressed and the lower jaw and supraorbital ridges unduly prominent, giving a grotesque appearance to the head. Corneal opacities and mental deficiency are also present.

**Cleido-cranial dysostosis (Fig 5 10)** This is a relatively rare condition in which there is aplasia of the clavicles with malformation of the bones of the skull and face.

**Other abnormalities** Local abnormalities of the head may be observed either in the nature of lumps or tumours (Figs 5 11, 5 12, 5 13, 5 14 and 5 15) or in the nature of *depressions* or indentations (e.g. depressed fracture of skull).

**Softening of skull (Craniotabes)** A curious softening of the bones of the skull in infancy allowing it to be indented with the fingers with the resilience of a ping-pong ball, and exhibiting "egg-shell crackling" is the commonest early sign of rickets disappearing before the end of the first year. It also occurs in osteogenesis imperfecta, hydrocephalus, congenital syphilis and hyperparathyroidism.

## FONTANELLES AND SUTURES

Normally, the anterior fontanelle closes about sixteen months, the posterior fontanelle about six weeks and the cranial sutures about six months after birth.

**Premature closure** of the cranial sutures is a characteristic feature of cranio-stenosis.

**Delayed closure** of the fontanelles and sutures is frequently noted in hydrocephalus, rickets, congenital syphilis and cretinism.

**Bulging fontanelles** in infancy suggest raised intracranial tension from meningitis, hydrocephalus or intracranial haemorrhage, they may be noted physiologically during crying.

**Sunken or depressed fontanelles** in infancy are indicative of dehydration or marasmus.

## THE HAIR OVER THE HEAD

**Colour of hair** This may be brown, black, ash-coloured, blonde or red. The colour depends on the size and number of melanin granules present, the presence or absence of a red pigment and on the thickness of the hair shaft.

**Curvature** Four varieties of curved hair are recognizable, viz (1) wavy hair, (2) helical hair with loops of constant size (as in Melaneseans and Europeans), (3) spiral hair, with diminishing sizes of loops distally (as in Negroes), and (4) peppercorn hair (with spiral hairs in clusters and knots)

**Greying of hair** This is due to loss of normal hair pigment and appearance of air bubbles in the hair-shafts. It is physiological after the age of 40 years, but may occur prematurely in some families (familial type), after severe mental shock and in generalized arteriosclerosis, coronary heart disease, pernicious anaemia or progeria (premature senility)

**Alopecia (Baldness)** *Diffuse hair loss* from the scalp may be due to (1) Acute physical (e.g. fever) or mental stress (e.g. bereavement) (2) Post-partum and with oral contraceptives (3) Iron-deficiency anaemia (4) Endocrine disorders—hypopituitarism, hypothyroidism, hyperthyroidism (rare), hypoadrenalism (5) Drugs—Thiouracils, anti-coagulants, excess vitamin A, antimitotic agents (6) Post-menopausal (7) Rapid weight loss (8) Idiopathic in majority. *Alopecia areata* (Fig. 5 16) is another common variety with patchy or circular areas of baldness, giving a characteristic "moth eaten appearance" to the head. It usually affects the temporal and occipital regions. The patches tend to be oval or circular in shape and sharply defined. There may be some short broken hairs which when removed show an attenuation of the bulb, thus tapering off from the thick shaft to the small knob of the bulb has given rise to the term 'exclamation mark' hairs. The cause of alopecia areata is unknown. Mental shock, emotional stress, focal infection, errors of refraction, endocrine disturbances and neuro-circulatory instability have been blamed.

Loss of hair from excessive friction or the use of strong soaps or shampoos is called *trichorrhexis nodosa*. Alopecia resulting from the habit of pulling out of hair is called *trichotillomania*. The hair in such cases may be ingested by mouth, resulting in a large "hair-ball" (trichobezoar) within the stomach.

The hair may become sparse or scanty in various conditions, e.g. fevers (such as typhoid), Simmond's disease, myxoedema, cirrhosis of liver, diabetes and syphilis. In myxoedema or hypothyroidism, the hair, besides being sparse, becomes coarse, dry and lustreless.

**"Bull dog" scalp** (Fig. 5 17) In acromegaly, the excessive growth of the skin and subcutaneous tissues of the scalp produces a thickened corrugated "bull-dog" scalp.

**Rheumatic nodules** These subcutaneous nodules may be seen on the back of the head in acute rheumatic fever (Fig. 5 18).

**"Syphilitic mop"** In syphilitic infants, there may be either abundant growth of hair over the head ("syphilitic mop") or extensive loss of hair.

## MOVEMENTS OF THE HEAD

These may occur normally in old age, in certain families or from the excessive use of alcohol or drugs, or in diseases such as Parkinsonism, aortic regurgitation, habit-spasm, Friedreich's ataxia, chorea, athetosis, multiple sclerosis and rickets.

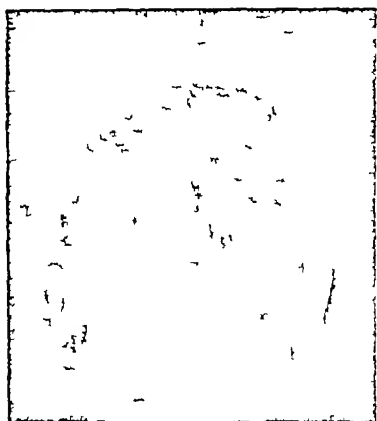


Fig 516 Alopecia areata in secondary syphilis



Fig 517 Bull-dog scalp of acromegaly

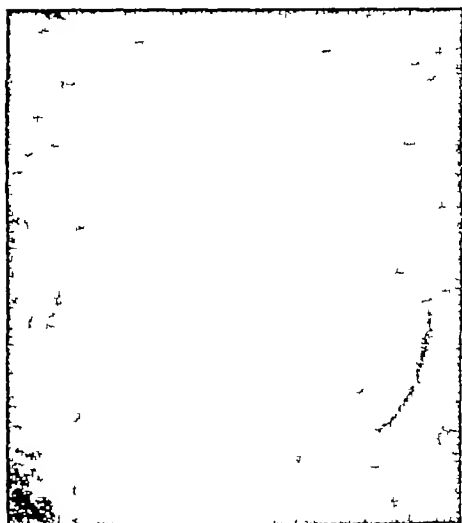


Fig 518 Rheumatic nodules on the back of head



Fig 519 Wrinkling of the forehead due to bilateral ptosis



Fig 520 Micrognathia with shrew mouse face





In *paralysis agitans* or Parkinson's disease, a slow, coarse and rotatory or nodding tremor or shaking of the head may be a constant and annoying symptom. It may be associated with a peculiar mandibular tremor (alternate opening and closing of the mouth) and a "trombone" (forward-backward) tremor of the tongue.

**Alfred de Musset's sign** Named after the great French poet and playwright, this sign consists of a constant forward and backward jerking of the head or visible movements of the ears, synchronous with the beating of the heart, in severe cases of aortic regurgitation.

In *habit-spasm* or *tic*, sudden jerky and repetitive movements of the head with or without facial "grimacing" movements may keep on recurring from time to time.

In *chorea*, unexpected, jerky and variable movements of the head may occur in conjunction with similar and better known "choreiform" jerky movements in other parts of the body, especially in children and young adults.

**Titubation** The phenomenon of irregular oscillation of the head and trunk, whilst standing, in cerebellar disease and in Friedreich's ataxia, is referred to as "titubation".

**Fixation of head** Lack of normal head movements with rigidity of the head and neck (cervical rigidity) may be due to meningitis or meningeal irritation, tetanus, strychnine poisoning, cervical spinal disease (such as caries, arthritis, neoplasm), trauma, cervical adenitis, extensive scars, torticollis or retropharyngeal abscess.

## THE FOREHEAD

**Prominence** Excessive prominence of the forehead or supraorbital ridges may be due to acromegaly, leontiasis ossium, enlarged air cells of the frontal sinuses, benign frontal exostosis (thickening of the frontal bone), tumours of the frontal bone (usually unilateral), chronic hydrocephalus, or to Parrot's nodes (frontal bossing).

**Wrinkling** *Bilateral wrinkling* of the forehead ("care-worn" or "wrinkled brow") may be suggestive of anxiety state, psychoneurosis or bilateral ptosis (Fig 5 19).

*Unilateral wrinkling* of the forehead may be due to facial paralysis (Bell's palsy).

*Absence of wrinkling*, bilaterally, on looking up is a useful sign of hyperthyroidism (Joffroy's sign).

**Local lesions** Local lesions of the forehead are both common and important, they may disfigure the general appearance of the patient.

*Scars* over the forehead are common in epileptics, who tend to fall from time to time with resultant injuries.

*Lumps* or *nodules* over the forehead may be post-traumatic or caused by tumours, gummata, osteomata, cysts or lesions of adenoma sebaceum (epiloia).

*Skin eruptions* are common over the forehead and may be due to acne, exanthematous fevers like small-pox, chicken-pox and measles, or to local skin diseases.

## THE FACE

### GENERAL APPEARANCE

Like the tongue or pulse, the facial appearance is a good index of underlying disease. Abnormalities of facial appearance may be due to alterations of contour, change in colour, affections of muscles or local lesions of the face.

**Congenital malformations** A featureless face from birth (*aprosopia*), an unduly small lower jaw (*micrognathia*) (Fig. 5.20), complete absence of the jaw (*agnathia*) and central eye with a proboscis (*cyclops*) have all been observed.

### CHARACTERISTIC TYPES OF FACIES

Many different types of facial appearance or facies have been described from time to time, with a view to facilitate recognition. In many cases, they are characteristic enough to permit an immediate or spot diagnosis of the causative disease. Of the diseases capable of producing characteristic types of facies, the most important are

- A. Diseases of the endocrine or ductless glands
  - (i) Pituitary: Acromegaly, Cushing's disease.
  - (ii) Thyroid: Cretinism, myxoedema, hyperthyroidism.
  - (iii) Gonadal: Eunuchoidism, bisexuality.
- B. Diseases of the nervous system: Parkinsonism, tabes, hemiplegia, facial paralysis, myasthenia gravis, myopathia.
- C. Infectious (acute and chronic): Acute exanthematous fevers, moribund states, congenital syphilis, tuberculosis, leprosy, tetanus.
- D. Cardiovascular diseases: Mitral stenosis, cyanotic heart disease, supraventricular aortic stenosis.
- E. Respiratory diseases: Lobar pneumonia, adenoids.
- F. Renal diseases: Nephrosis.
- G. Avitaminosis: Ariboflavinosis.
- H. Congenital anomalies: Mongolism, hypertelorism.
- I. Miscellaneous causes: Moon face, scleroderma, cirrhotic facies.

### CLINICAL DESCRIPTIONS OF COMMON TYPES OF FACIES

#### *Of Endocrine Origin*

**Acromegalic face** The face in acromegaly displays an ape-like appearance (Fig. 5.21) with a prominent lower jaw (prognathism), coarse features, large nose, lips and ears, prominent forehead and cheek bones, and widely spaced teeth.

**Face in Cushing's syndrome** A basophil adenoma of the pituitary, adrenal cortical tumour, adrenal cortical hyperplasia or tumour of the ovary or thymus may be characterized by a "moon-face" or face rounded by obesity, a flushed or plethoric facial skin with acne, and excessive growth of hair (*hirsutism*) (Figs 5.22, 5.23).



Fig 5.21 Acromegalic face



A



B

Fig 5.22 Cushing's syndrome A Face before onset of disease B When characteristics of Cushing's syndrome were manifested (Courtesy Dr P N Shah, Indian Cancer Research Centre, Bombay)



Fig 5.23 Cushing's syndrome in a boy aged 2 years Note plethoric moon-face, oily skin and acne A non-malignant tumour of the right adrenal cortex was found (Courtesy Dr P N Shah Indian Cancer Research Centre, Bombay)

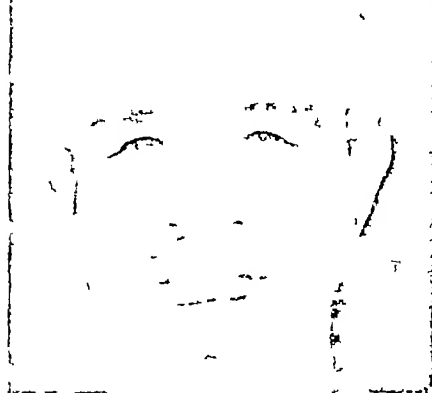


Fig. 525 Myxoedematous face

Fig. 526 Hyperthyroid face

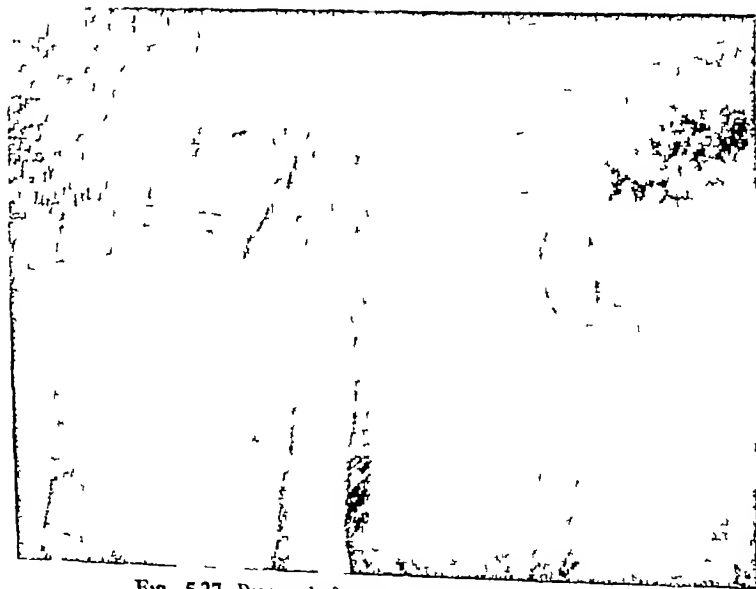


Fig. 527 Bisexual face in pseudohermaphrodite  
Note the genitalia

**Cretinoid face** (Fig 5 24) In cretinism, the face is pale with a stupid, dull or somnolent appearance, the skull and face appear wide, the nose broad and flattened, lips thick and separated by a large, fissured and protruding tongue, and the eyebrows, eyelashes and hair on the head scanty and dry

**Myxoedematous face** (Fig 5 25) In myxoedema, the face is pasty, pale and puffy, the expression dull, apathetic and emotionless and the eyelids swollen and drooping, there is loss of hair over the eyebrows and head and the skin is thick and dry with a characteristic malar flush

**Hyperthyroid face** (Fig 5 26) In thyrotoxicosis, the facial expression is one of fright, anxiety or alertness, the eyes are prominent, shiny and staring, the skin is moist, and emotional movements of the face excessive

**Eunuchoid facies** In male hypogonadism, the facial skin is sallow and pale, with wrinkles or furrows and absence of facial hair. The eyes may appear mongoloid from deposition of fat in the upper lids

**Bisexual face** (Fig 5 27) The development of manly characteristics (such as a beard or masculine type of nose or jaw) in a female, or the lack of facial hair in a male, is observed in the so-called bisexual face

**Infantile facies or juvenile facies** in an adult is suggestive of hypogonadism or hypopituitarism. The opposite condition of *premature ageing*, or elderly appearance in a youthful subject, is occasionally encountered in some families as an inherited trait and at times in progeria, generalized or coronary atherosclerosis and in Simmond's disease

### *Of Neurological Origin*

**Mask-like face** (Parkinsonian mask) An immobile, fixed and expressionless face (with the head and neck moving as a whole, owing to rigidity of muscles) is typical of the Parkinsonian syndrome

**Tabetic facies:** The association of drooped eyelids or partial ptosis with wrinkling of the forehead (from over-action of the occipitofrontalis muscle) and unequal small and irregular pupils, suggest a diagnosis of tabes dorsalis. The facial expression is said to be one of "sadness"

**Apoplectic facies** After a cerebrovascular accident or "stroke", with resultant hemiplegia, the patient may display a pale or plethoric, expressionless countenance with saliva drooping from one angle of the mouth, and thick and laboured speech

**Asymmetrical face** An asymmetry of the face or inequality of the two halves of the face may be due to unilateral paralysis of the facial nerve (Bell's palsy) or congenital hemiatrophy of face (Fig 5 28)

**Myasthenic facies** (Fig 5 29) There is a characteristic drooping of the upper eyelids and chin, with ptosis and open mouth, particularly noticeable

towards evening. In some cases, the most noticeable feature is a peculiar "sneering smile" or "myasthenic sneer" due to weakness of the zygomatic and risorius muscles

**Madonna-like face** In the facio-scapulo-humeral myopathy of Landouzy and Dejerine, the face is described as "madonna-like" with a "transverse smile"

**Myopathic facies** (Fig 5 30) In neuromuscular disorders involving the facial muscles, the facial appearance is often characteristic with a loose pouting of the lips exposing a large part of the labial mucosa, a peculiar transverse smile, and either drooping of the upper eyelids or difficult closure of the eyes

### *Of Infective Origin*

The facies in *exanthematous fevers* (Fig 5 31) and *acute illnesses* may be characteristic enough to suggest an immediate diagnosis. A peculiar rounded contour of the face, from bilateral enlargement of the parotid glands, is common in mumps and (rarely) in Mikulicz's syndrome (Fig 8 32) where the lacrimal glands are also markedly enlarged. The eruptions of measles, chicken-pox, small-pox and scarlet fever may be easily observed and recognizable over the face. In scarlet fever, there is a diffuse bright-red rash with circumoral pallor. In measles, the morbilliform rash, congested and watering eyes and heavy nasal cold are easy to recognize. In trichinosis, the eyelids may be swollen and drooping.

**Hippocratic facies** First described by Hippocrates, this sign is of evil omen. The sunken and dull eyes, pinched nose, parched and dark skin of the face, with a tendency to sink in bed, muttering delirium, bed-picking (carphology) and dance of the muscle tendons (subsultus tendinum) are typical of a very serious, preterminal condition, sometimes referred to as the "typhoid state" or toxic state.

**Congenital syphilitic face** (Fig 5 33) A characteristic type of face with sunken bridge of nose (saddle-nose), rhagades and interstitial keratitis is suggestive of congenital syphilis.

**Tuberculous (or phthisic) facies** The face appears thin, wasted and pale, with or without a hectic flush over the cheeks. The eyelashes are frequently long and silky and the skin delicate.

**Leonine face** (Fig 5 34) In leprosy, the thickening and superabundance of skin together with a flattened nose and loss of hair of the eyebrows and eyelashes results in a leonine facies.

**Face in tetanus** Spasm of the facial muscles in the early stage of tetanus may lead either to retraction of the angles of the mouth with a peculiar sardonic smile (risus sardonicus) (Fig 5 35) or to a pursing of the lips. The eyes may be partly closed (contraction of orbicularis oculi) and the eyebrows elevated (contraction of frontalis muscle) (Fig 5 36).



Fig 5.28 Congenital hemiatrophy of face

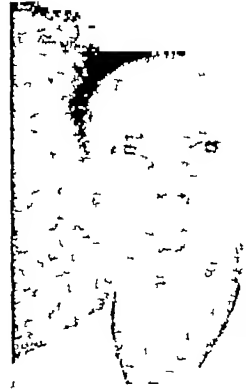


Fig 5.29 Myasthenic face  
A Before neostigmine B After neostigmine

Fig 5.30 Myopathic facies

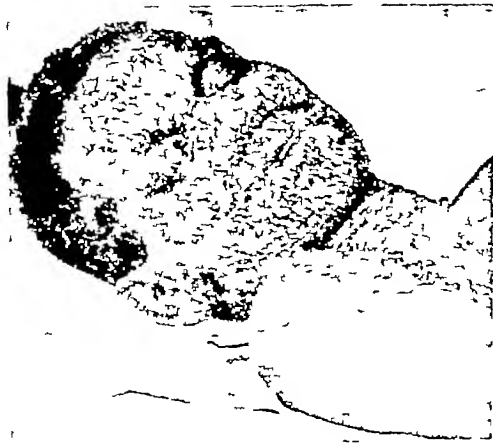


Fig 5.31 Face in confluent smallpox



Fig 5.32 Mikulicz's syndrome





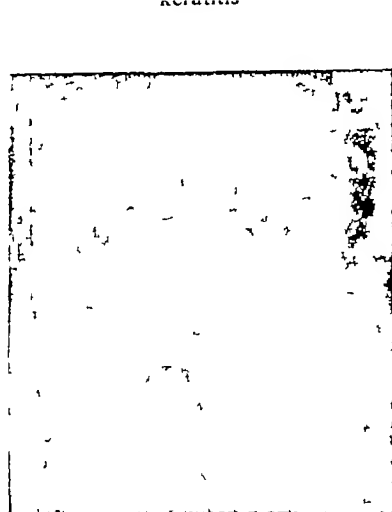
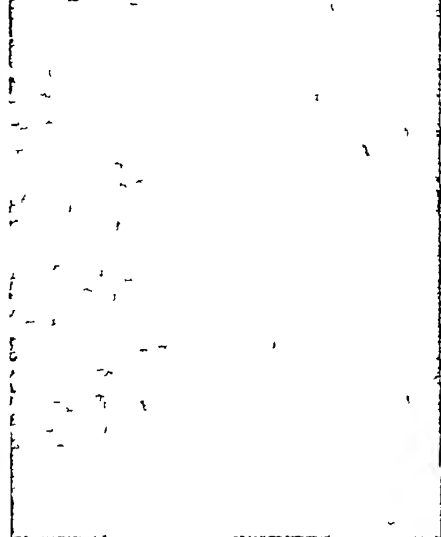


Fig. 5.34 Leonine face of leprosy

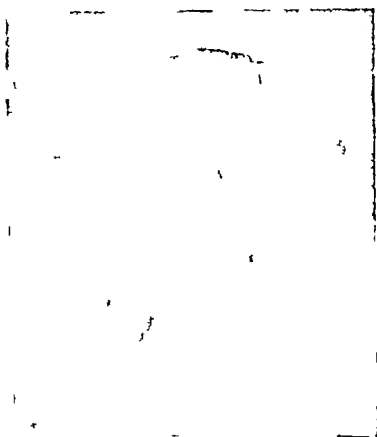


Fig. 5.35 Risus sardonicus of tetanus



Fig. 5.36 Face in cephalic tetanus

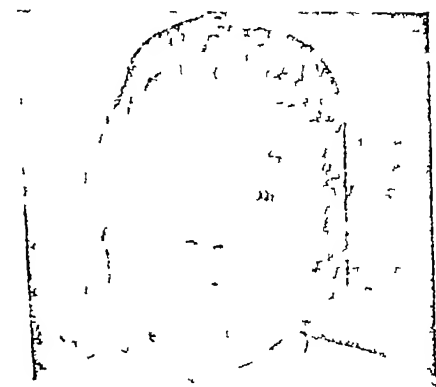


Fig. 5.37 Nephritic facies of acute nephritis

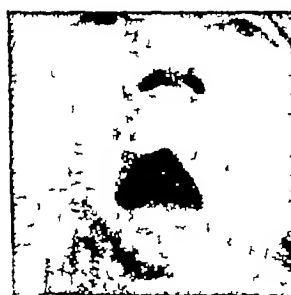


Fig. 5.38 Aribosflavinosis with angular cheilitis

### *Of Cardiovascular Origin*

**Mitral facies** (Malar flush) In mitral stenosis, venocapillary stasis causes a bluish tint or flush in the prominent areas of the face such as cheeks, nose and ears. Mitral facies occurs only in chronic disease with high pulmonary vascular resistance.

**Cyanotic facies** Cyanosis of the head and neck is usually part of a generalized cyanosis. In cyanotic congenital heart disease, the cyanotic hue of the lips, nose and ear lobes may be particularly striking. In superior vena caval obstruction (from aortic aneurysm, mediastinal tumour, constrictive pericarditis), the face, neck and arms show congestion with oedema, the congestion increasing on stooping.

**Elfin-like facies** In supravulvular aortic stenosis there is a wide mouth with large lower lips (pouting effect), widely spaced teeth, broad forehead, pointed chin, heavy cheeks and protruding ears, and eyes set well apart.

**Rounded face** In congenital pulmonary stenosis the cheeks are often high coloured and the face rounded.

### *Of Respiratory Origin*

**Pneumonic facies** In lobar pneumonia, one or both cheeks may show a malar flush, the alae nasi appear distended and overactive, the eyes bright and shiny, and herpetic lesions may be visible over the lips.

**Adenoid facies** In children with massive adenoids, the mouth remains continuously open, with a pinched nose, conferring a somewhat "stupid" expression.

### *Of Renal Origin*

**Nephrotic (Nephritic or Renal) facies** (Fig 5 37) The face appears puffy, pale and swollen, with baggy eyelids and narrowed palpebral fissures. These characteristics are most obvious during the morning hours.

### *Of Deficiency Type*

**Arriboflavinosis face** (Fig 5 38) In vitamin B<sub>2</sub> or riboflavin deficiency, the face may show angular cheilitis with superficial fissures, scars or yellow crusts at the angles of the mouth, seborrhoic lesions of the facial skin, mild erythema with scaly desquamation over the alae nasi and ears, and conjunctivitis with red itchy and half-closed eyes from photo-phobia.

### *Of Congenital Origin*

**Mongoloid facies** In Mongolian idiocy, the face is characterized by slanting almond-shaped eyes (Fig 5 39), presence of epicanthic folds, large ears, broad and flat nose, florid coarse tongue, scanty and wiry hair on the head and an amiable but stupid expression.

**Hyperteleorism** (Hereditary craniofacial dysostosis) (Fig 5 40) In this interesting condition, sometimes associated with congenital heart disease, the eyes are set wide apart with a depressed nasal bridge, a low forehead with a vertical groove, prominent frontal bossing, optic atrophy and mental retardation. The queer shape of the forehead is due to congenital enlargement of the lesser wings of the sphenoid bone.

**Fish-like facies** In incomplete mandibulo-facial dysostosis there is an anti-mongoloid slant of the eyes, hypoplasia of malar bones and mandible, macrostoma and sunken cheek bones.

### *Of Miscellaneous Origin*

**Moon-face** A face rounded by oedema or fat is sometimes referred to as "moon-face". It has been described in renal diseases, such as nephrosis and acute nephritis, in Cushing's syndrome (Figs 5 28, 5 29) and after cortisone or ACTH therapy. The mouth may be shaped like a "fish-mouth" with corners being depressed when the patient is obese.

**Scleroderma face** (Fig 5 41) The facial skin may be so markedly tightened in scleroderma that smiling and proper closure of the mouth are prevented. The smooth skin, partially open mouth, exposed teeth and fixed smile suggest the diagnosis.

**Cirrhotic facies** In early cases of cirrhosis from alcoholism, the face is sallow and bloated or flushed and florid, with injected and watering eyes, prominent pink nose and dilated vessels over the cheeks and nose. In the later stages, the eyes and cheeks become sunken, the eyes icteric and congested and the face and neck covered with spider angiomas.

**Facial deformities** Bizarre deformities of the face may result from a variety of conditions, such as new growths arising from the bones or soft tissues of the face (Fig 5 42) or long-standing facial paralysis (Fig 5 43).

**Bull-dog face** In pseudoxanthoma elasticum the skin over the mouth may sag giving a bulldog-like appearance.

**Weeping face** When facial muscles are affected in dermatomyositis, there is a violaceous hue with oedema of the upper eyelids and a weeping facial expression is observed.

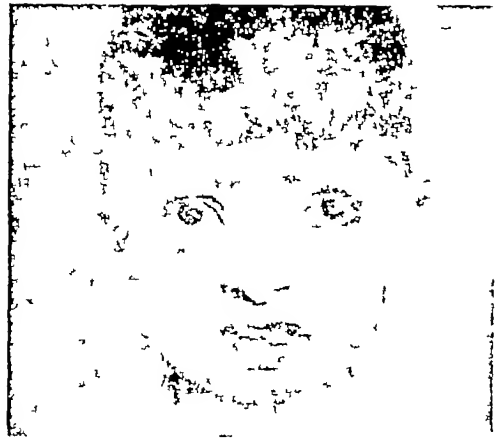
**Coarse facies** In pachydermoperiostosis with idiopathic clubbing the skin over the face and forehead is thick and oily (Fig 5 44).

### COLOUR OF FACE

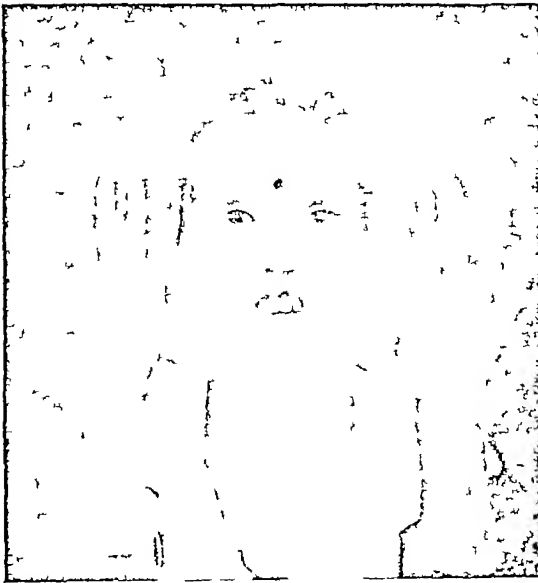
Significant alterations in colour of the skin are frequently appreciated better over the face than elsewhere and should always be looked for with care. Inspection of the skin should always be carried out in broad daylight rather than



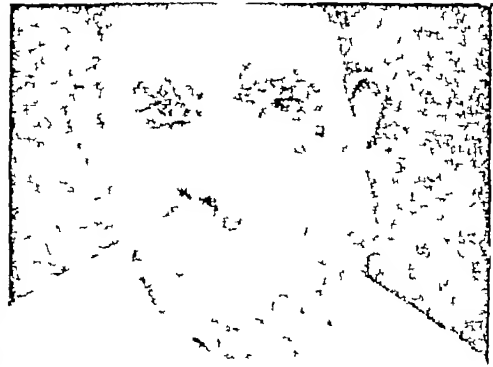
**Fig 5.39** Microcephaly with mongolism



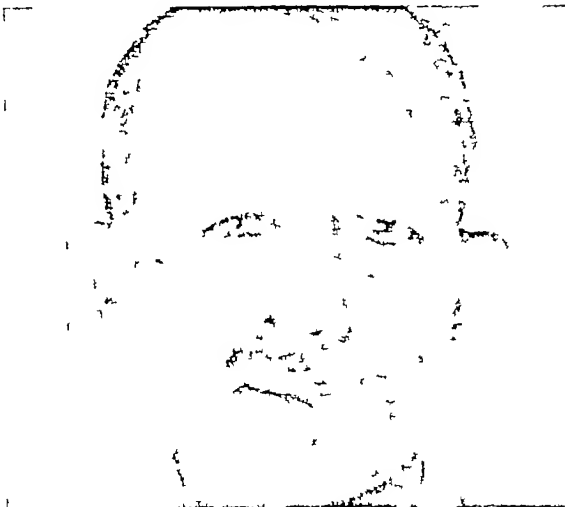
**Fig 5.40** Hypertelorism



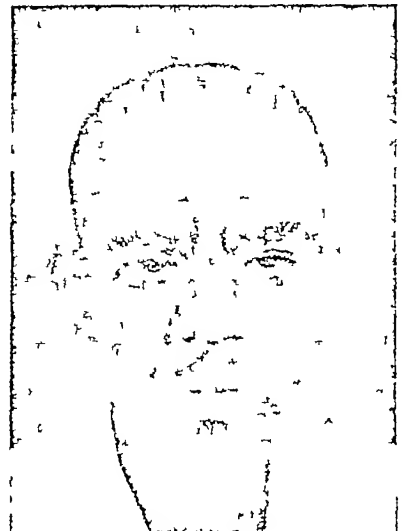
**Fig 5.41** Scleroderma face



**Fig 5.42** Deformity of face owing to cavernous hamangioma



**Fig 5.43** Facial asymmetry owing to contracture from chronic facial palsy



**Fig 5.44** Coarse facies of pachydermoperiostosis



**artificial light** The most important changes of colour to note, over the face, are ruddiness or plethoric appearance, pallor, cyanosis, jaundice and decrease or increase of skin pigmentation

**Ruddy complexion or flushed face** This may be *physiological* and due to constant exposure to the sun and air ("weather-beaten" appearance), over-eating, chronic indigestion, alcoholism, cold climate, high altitude or the use of artificial aids to beauty *Pathological* ruddiness of face may be due to polycythaemia vera or acquired polycythaemia, febrile conditions, acute lobar pneumonia, cerebral apoplexy, vasomotor disturbances, climacteric syndrome, acute pulmonary tuberculosis (hectic flush), mitral stenosis (mitral flush), acne rosacea, lupus erythematosus, or erythematous rash over the face *Transient* or *evanescent* flushing of the face is common in emotional states of shyness, anger or fatigue, particularly in girls It may occur in carcinoid syndrome and in patients on oral antidiabetics especially chlorpropamide after alcohol intake

**Facial pallor**, usually part of a generalized pallor of the skin, may be *physiological* (as in some families and individuals), or *climatic* (after long residence in the tropics) Different degrees of facial pallor may be due to anaemia A pale face is often observed in acute rheumatic carditis, sub-acute bacterial endocarditis, malignant hypertension, advanced stages of insufficiency and stenosis of the aortic valve, in state of extreme precordial pain as in angina pectoris, and in peripheral vascular collapse or cardiogenic shock

**Jaundice** may be apparent on the face earlier than elsewhere The yellow or yellowish-green discoloration of the sclerae, an early and important sign of jaundice, serves to distinguish the latter from other causes of yellow discoloration of skin (such as carotinaemia)

**Cyanosis of face** Although usually generalized or extensive, cyanosis is often more obvious over the face (especially over the lips, cheeks and ear-lobes) and nails than elsewhere

**Depigmentation** Albinism and leucoderma are frequently recognizable by a look at the face In albinism, the glistening white skin, colourless hair and pink iris are characteristic enough to suggest the diagnosis at once In leucoderma, the face may display typical areas of depigmentation with sharp margins and surrounding areas of hyperpigmentation

**Excessive pigmentation** The skin of the face may show excessive pigmentation in a variety of conditions (Fig 5 45) The dark brown pigmentation of the facial skin and buccal mucosa in Addison's disease, the bronzed or slate-grey appearance of the skin in haemochromatosis, the greyish-black or greyish-blue pigmentation of argyria, the yellowish-brown patches of chloasma on the forehead and face in pregnancy ("mask of pregnancy") and the bluish appearance of the ear-cartilages in ochronosis are characteristic enough, when seen on the face, to suggest the right diagnosis at once

**Sweating of face** This may be part of a generalized sweating (hyperidrosis), or localized to one or both sides of the face or to a part of the face only. Localized sweating of the face may be observed in trigeminal neuralgia, syringomyelia, post-encephalitic Parkinsonism, granulosus rubra nasi (redness and sweating confined to the nose), local injuries and in some normal individuals. Asymmetrical gustatory hyperidrosis refers to localized sweating of the face, head and neck on one side after eating or drinking (usually attributed to the destruction of the preganglionic sympathetic fibres involved in sweating).

**Facial spasm** Spasms of the facial muscles may be tonic or clonic, localized or diffuse, unilateral or bilateral. The important causes of facial spasm are Bell's palsy, trigeminal neuralgia, lesions of the seventh nerve nucleus, epilepsy, chorea, hysteria, tetanus, meningitis, encephalitis, thyrotoxicosis, habit spasm, mimic spasm (in adults), convulsive tic, blepharospasm and nictitating spasm.

**Facial pain** Neuralgic or neuritic pains are very common over the face, the important types being (1) *trigeminal* neuralgia (tic douloureux), with intense, paroxysmal, darting pains radiating from the lip or nose to the side of the face, forehead or eye on one side, in elderly subjects, (2) *sphenopalatine* neuralgia, with continuous, boring pain in cheek bone, gums, palate and neck, (3) *glossopharyngeal* neuralgia with paroxysmal darting pain from the tonsillar fossae to the ear, promoted by talking, deglutition or yawning, (4) *dental* neuralgia, (5) *post-herpetic* neuralgia, and (6) *sinusitis* pain.

## THE EYES

### THE EXPRESSION

The expression of the eyes may reflect the personality of the individual and his reaction to disease. To the observant physician, it may afford information of value.

The expression of the eyes may be dull, apathetic or miserable in cases of depressional psychosis, melancholia, myxoedema and cretinism. A wild expression is encountered in mania, mental derangement or bad temper. Shiny, bright or staring eyes are common in hyperthyroidism, typhoid fever, pneumonia and neurosis. A blank or distant expression is characteristic of schizophrenia, coma or clouding of the consciousness. A shiftness or furtiveness of the eyes may suggest undependability, untruth or malingering.

### THE EYEBROWS

In myxoedema, there is a characteristic loss of hair over the outer third or more of the eyebrow on either side. In the secondary stage of syphilis, the loss of hair is patchy or uneven. In leprosy, the skin and subcutaneous tissues underneath the eyebrows may show thickening or superabundance, an early sign of the disease.

## THE EYELIDS

**Ptosis** Ptosis or drooping of the upper eyelid with an inability to open the eye is a fairly common finding. It may be unilateral or bilateral, partial or complete, congenital or acquired, constant or variable, transitory or permanent, and isolated or associated with other neurological manifestations. The important causes of ptosis are (1) paralysis or involvement of the oculomotor nerve, its nucleus or cortical centre (leading to paralysis of the levator palpebrae muscle), the ptosis is unilateral, complete and associated with a squint and large pupil, (2) involvement with paralysis of the cervical sympathetic nerve (Horner's syndrome), the ptosis is usually unilateral, partial and with a small pupil, (3) myasthenia gravis, where the bilateral ptosis is transitory and develops towards evening from muscle fatigue, (4) ocular myopathy, (5) marasmus, debility or dehydration, (6) oedema, tumour or trachoma of the eyelid causing increase in the weight of the lid, (7) tabes dorsalis (pseudoptosis with furrowing of the brow and pathological pupils), (8) encephalitis lethargica, (9) acute infectious polyneuritis, (10) botulism, (11) tetanus, (12) hysteria, (13) trichiniasis, (14) trigeminal neuralgia, (15) cerebral ptosis (isolated ptosis), and (16) congenital ptosis (due to abnormal superior rectus muscle).

**Oedema of eyelids** Puffiness or swelling of the eyelids, especially the lower ("baggy eyes"), may be *physiological* and due to lack of sleep, cough or excessive crying, or *pathological* and due to nephritis or nephrosis, anaemia, hypoproteinaemia, beri-beri, allergic states, hay fever, angioneurotic oedema, trichinosis, whooping cough, measles, small-pox, chicken-pox, iodism, neurosis, superior vena cava obstruction, cavernous sinus thrombosis, diabetes, intracranial lesions, insect bites or local eye diseases.

**Mikulicz's syndrome** In this disease, the enlargement of the lacrymal glands is responsible for the characteristic bilateral swellings of the upper eyelids. An associated bilateral enlargement of the parotid glands makes the diagnosis certain. (Lacrymal glands may enlarge in sarcoidosis).

**Hordeolum or sty** An infected follicle or small abscess in the upper or lower eyelid is a fairly common condition, frequently due to general debility, eye-strain, refractive error or bad lighting.

**Chalazion (Meibomian cyst)** A slow growing tumour or cyst of the meibomian gland in the eyelid, distinguishable from the familiar "sty".

**Dacrocystitis** Inflammation and swelling of the lacrymal sac at the inner canthus of the eye. Acute and chronic forms are recognized. A chronic cyst, secondary to chronic inflammation, is called a mucocele.

**Xanthomata (Xanthelasma palpebrum)** These are single or multiple, unilateral or bilateral, raised or flattened, soft, yellowish or orange coloured nodules or plaques from 1 mm to 1.5 cms in diameter, visible and palpable over the eyelids (especially over the inner ends of the upper eyelids) or in their close vicinity. They are common in old age and in cases of diabetes, coronary heart disease and generalized arteriosclerosis. Sometimes familial, this condition is due to deposition of fat.



**Tophi** In gout, tophi containing urate crystals may occasionally arise over the eyelids

**Coloboma** A notch in the upper eyelid at the junction of the middle and inner third

**Ectropion** Eversion of free edge of the eyelid may be due to old age, facial paralysis (with orbicularis oculi involvement) or to scarring of the lids **Entropion** or rolling-in or inversion of the eyelid may be due to spasm of the orbicularis oculi (*spastic* type) or to scarring (*cicatricial* type) **Trichiasis** or ingrowing of the eyelashes with corneal irritation is common in trachoma

**Blepharospasm** The term has been indiscriminately applied to both increased blinking of the eye and a narrowing of the palpebral slit.

**Blepharitis** Inflammation of the eyelids with thick, red, angry and encrusted margins may occur in measles, hay fever, anaemia, eye infections or idiosyncratically

**Herpes zoster ophthalmicus** Small groups of vesicles on an erythematous base may be noted on the eyelid (later to be replaced by pigmented scars) when the ophthalmic division of the trigeminal nerve is involved in herpes zoster

**Basal-cell epithelioma** This variety of carcinoma ("rodent ulcer") is fairly common over the lower eyelid in elderly subjects and usually takes the form of a slow-growing ulcer with indurated margins

**Blinking** Blinking of the eyelids may be unduly or embarrassingly frequent in habit-spasm (tic), in nervousness or local eye irritations Infrequent blinking (Stellwag's sign) is frequently observed in cases of Grave's disease and Parkinsonism Normally, blinking occurs about 3 to 5 times per minute A tremor or flickering of the eyelid on closing the eye is a common finding in cases of Parkinsonism

**Myokymia** A transient quivering or flickering of the orbicularis oculi muscle ("live flesh"), so-called myokymia, is common in anaemia and general debility and is of no significance

**Lagophthalmos** Inefficient or incomplete closure of the eye, which may lead to conjunctivitis or corneal ulceration, may result from weakness or paralysis of the orbicularis oculi muscle (as in Bell's palsy), exophthalmic goitre, orbital tumour with proptosis, during convulsions, hysteria, moribund patients, just after death, after scarring of the eyelids or as a congenital abnormality

**Asynergia** of the eyelid (oculopalpebral asynergia) or delayed downward movement of the upper eyelid on looking downwards (von Graefe's sign) is typical of Grave's disease

#### PALPEBRAL FISSURE

**Normal variations** The palpebral fissure normally tends to become narrower with age Racial differences are quite noticeable, the palpebral fissure being widest in the whites, of moderate size in Negroes, and narrow, slanting and obliquely placed ("almond-shaped" eyes) in Mongolian races In the last, there is a characteristically prominent epicanthic fold (Mongoloid fold) due to an abnormal direction of the marginal fibres of the orbicularis oculi muscle

**Abnormalities of size.** The palpebral fissure may become unduly wide, narrow or even obliterated in disease. A *wide* palpebral fissure is noted in facial nerve paralysis, cervical sympathetic stimulation (opposite of Horner's syndrome), hyperthyroidism (Dalrymple's sign) and Parkinsonism. A *narrow* palpebral fissure is customary in ptosis or drooping of the upper eyelid (from third nerve paralysis, Horner's syndrome, myasthenia gravis, etc.) and in photophobia, cerebral irritation and local eye conditions. In the latter states, the eyes may be screwed up or tightly closed with complete obliteration of the palpebral fissure. Obliquely placed or *slanting* palpebral fissures ("almond-shaped") are a characteristic feature of mongolism or mongolian idiocy.

**Lacrymation.** The state of moisture of the eye may afford useful information. *Excessive lacrymation* or secretion of fluid may be physiological as in crying or laughing or pathological and due to a foreign body in the eye, local inflammation of the eye, measles, influenza, whooping cough, hay fever, coryza, typhus, facial palsy, trigeminal neuralgia, iodism, irritant vapours or eye-drops and exposure to inclement weather. *Epiphora* or overflow of tears from the eyes may be due to any of the causes of excessive lacrymation, to affections of the lacrymal canal or to old age. *Dryness* of the eyes or defective or absent secretion of fluid may arise in xerophthalmia, trachoma, lagophthalmos or long-continued fevers.

**Dark rings.** Circles around or under the eyes (duskiness of the eyes) are of no pathological significance they being frequently associated with tiredness, general debility, menses, indigestion, lack of sleep, pain of malignancy. They may occur as a hereditary or familial characteristic, especially in brunettes with pale skins.

**Black eye.** The bluish, purplish or dark brown discoloration of the skin in the familiar "black eye" from a blow or injury is due to the blood which oozes into the loose subcutaneous tissues of the eyelid showing through the thin and transparent skin of the lid.

## THE EYEBALLS

The eyeballs may be either protruded forwards and prominent (*exophthalmos* or *proptosis*) or retracted, indrawn or sunken (*enophthalmos*). In either case the condition may be *bilateral* or *unilateral*.

**Bilateral exophthalmos** is most frequently associated with hyperthyroidism (Grave's disease) (Fig 5 26) but also occurs in oxycephaly (turret skull) (Fig 5 5), severe myopia, chronic cor pulmonale (frog's eyes) (Fig 5 46), severe dyspnoea or asthma, subperiosteal haemorrhages from scurvy, leukaemias, neurasthenia, thrombosis of the superior longitudinal sinus and rarely in normal individuals as a congenital anomaly. In hyperthyroidism, the exophthalmos, which may at times start unilaterally, is at times severe enough to prevent eyeball movements upwards and outwards (exophthalmic ophthalmoplegia) or even to cause complete dislocation and destruction of the eyeball (malignant exophthalmos) (Fig 5 47). Usually, however, the condition is of a milder order and gives the patient a "frightened" or "terror-stricken" appearance, with sclera visible all around the iris.

The immediate cause of exophthalmos in thyrotoxicosis is a quantitative increase in the bulk of the retro-orbital tissues from oedema and deposition of fat in the connective tissues and eye muscles. The remote cause is uncertain but experimental evidence suggests that the

pituitary may play a significant part Exophthalmos is recognized by exposure of the sclera below the iris Retraction of the upper eyelid is perhaps more frequent than exophthalmos and can be recognized by exposure of the sclera above the iris

**Unilateral exophthalmos** may be observed in early cases of Grave's disease, or in cellulitis or tumours within the orbit (Fig 5 48), orbital haemorrhage (as in scurvy or purpura), sinusitis, leukaemia, xanthomatosis (Hand-Schuller-Christian syndrome), meningioma, acromegaly (from bony overgrowth), high myopia, arteriovenous aneurysm (Fig 5 49), varicose ophthalmic veins and cavernous sinus thrombosis

**Pulsating exophthalmos** (almost invariably unilateral) is a rare but characteristic entity observed in arteriovenous aneurysm or fistulous connection between the internal carotid artery and cavernous sinus (usually resulting from trauma or miliary aneurysm formation)

**Enophthalmos** Retraction, recession or sinking of the eyeball in its socket occurs in states of dehydration or shock (as in cholera, dysentery and gastroenteritis), long continued fevers, wasting or cachexia, and in moribund patients (with Hippocratic or typhoid facies), the condition being bilateral Unilateral enophthalmos occurs in Horner's syndrome or sympathetic paralysis (associated with ptosis and small pupil) and after injury or haemorrhage involving the orbit Unilateral or bilateral enophthalmos may also be a congenital anomaly

**Tension of eyeballs** Undue hardness or softness of the eyeball on digital palpation may be indicative of disease Undue hardness of the eyeball is suggestive of increased intraocular tension as in glaucoma Undue softness ("jelly-like"), on the other hand, occurs in dehydration and diabetic coma

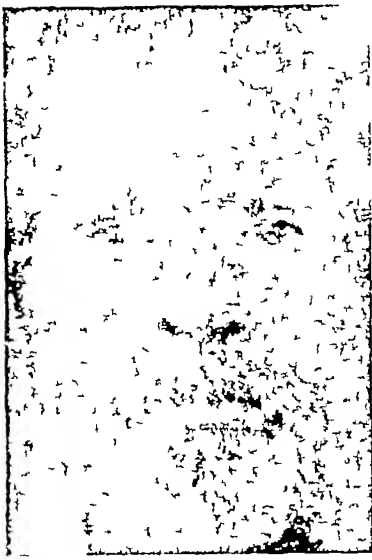
**Visible pulsation** of one or both eyeballs may be noted in cases of arteriovenous aneurysm, aortic regurgitation, vascular tumour, hypertension or arteriosclerosis

**Skew deviation** In cerebellar tumour or injury, the eyeball on the affected side may be deviated downwards and inwards, whilst the contralateral eyeball looks upwards and outwards, a phenomenon referred to as "skew deviation"

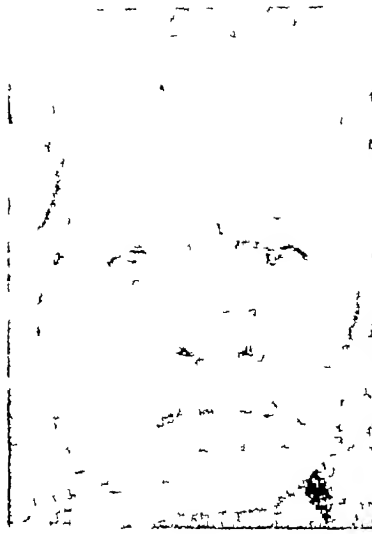
**Fixed eyeballs** Weakness or paresis of extrinsic eye muscles causing complete or partial fixation of the eyeballs and lack of eyeball movements (external ophthalmoplegia) may occur in exophthalmic goitre and in neurological disease

**Wandering eyeballs** A wandering of the eyes, each eye moving apparently independently of the other, may be noted in moribund patients and in meningitis ("chameleon movements") The movements of the eyeballs are usually slow, involuntary, dissociated and lateral

**Oculogyric crisis** (Oculogyric spasm) Sudden paroxysmal, tonic, upward deviation of both eyeballs for seconds or minutes may be observed in the post-encephalitic form of Parkinsonism



A



B

Fig 545 Pigmentation of  
Pellagra  
A Before treatment.  
B After treatment



Fig 546 Frog's eyes in chronic  
cor pulmonale



Fig 547 Malignant exophthalmos



Fig 548 Unilateral exophthalmos from orbital tumour



Fig. 549 Unilateral exophthalmos with  
congestion of the eye owing to left-sided  
traumatic carotico-cavernous fistula



**Conjugate deviation** Persistent and pronounced turning of both eyes to one or other side (due to associated spasm or paralysis of the internal rectus muscle on one side and the external rectus on the other) may be seen in cases of hemiplegia. The deviation is toward the hemiplegic side in brain stem lesions and away in cortical lesions.

**Doll's eyes movements (Doll's head phenomenon)** This depends on the normal vestibular reflex mechanism which enables ocular fixation to be maintained despite head movement. In absence of damage to the brain stem, rolling of the head in a drowsy or semiconscious subject leads to conjugate deviation of the eyes to the other side. Neck flexion produces upgaze, and extension downgaze. In deep coma of whatever cause these movements can be difficult to elicit.

**Moebius's sign** A failure of convergence of the eyeballs on fixing a near object may be noted in cases of hyperthyroidism (*Moebius's sign*) and post-encephalitic Parkinsonism.

**Heterophoria** Weakness (asthenopia) or tiredness of the ocular muscles after intensive use of the eyes, resulting in a tendency to deviation of the ocular axis (without a frank squint), either in an outward (exophoria), inward (esophoria), upward (hyperphoria) or downward (hypophoria) direction. It may cause headache, migraine, or vertigo in susceptible subjects.

**Panophthalmitis** Infection of the eyeball, which may be secondary to wounds, ulcers or operation (exogenous type) or to septicaemia or pyaemia (endogenous type).

**Sympathetic ophthalmia** A rare condition, commoner in childhood, where injury to one eye is followed after a few weeks by serious inflammation (even with loss of sight) in the opposite eye.

## THE CONJUNCTIVA

The conjunctiva comprises of the palpebral conjunctiva underlining the eyelids and the bulbar or ocular conjunctiva lining the front of the eyeball.

**Conjunctivitis** Inflammation of the conjunctiva is a fairly common entity characterized by itchiness or burning of the eye and bright red coloration of the conjunctiva associated with lachrymation and discharge.

**Petechial lesion** Minute pin-point haemorrhages of the conjunctiva are an important sign of subacute bacterial endocarditis but may occur also in diseases of the blood (e.g. leukaemias and aplastic anaemia).

**Subconjunctival ecchymoses** Large subconjunctival or conjunctival haemorrhages may be encountered in hypertensive states, after severe bouts of coughing (as in whooping cough), straining or vomiting, during convulsions or fits, in violent dyspnoea, after fracture of the base of the skull and in the course of pyaemia, septicaemia or subacute infective endocarditis.

**Pterygium** A fold of membrane which extends from the outer part of the bulbar conjunctiva to the cornea, usually in elderly persons exposed to dusty air.

**Symblepharon**. A scarlike attachment, usually post-traumatic, between the palpebral and bulbar conjunctiva.

**Bitot's spot** A white or greyish plaque with a foamy surface, close to the limbus, on the exposed area of bulbar conjunctiva and usually near the temporal rather than nasal aspect. The plaque is caused by desquamated epithelial cells. Usually found in vitamin A deficiency, Bitot's spots have been known to be due to local eye irritations of various kinds.

## THE SCLERA

The firm, fibrous outer coat of the eyeball is called the sclera or sclerotic.

**Yellow sclerotics** A yellow or yellowish-green discoloration of the sclerotics is an early and important sign of jaundice or icterus, especially in dark-skinned individuals in whom the icteric tinge of the skin is frequently obscured by the skin pigmentation.

*Normal colour* of the sclerotics in the presence of yellow skin definitely rules out jaundice as the cause of the yellow colour, the possibility of a carotinaemia should be considered in such a case.

*Patchy yellowness* of the sclerotics is suggestive of subconjunctival fat, and is common in pernicious anaemia.

**Scleritis** A rare condition of inflammation of the sclera with severe pain, secondary glaucoma and red or violet spots near the cornea.

**Staphyloma** A condition of bulging with local thinness of sclerotic coat.

**Blue sclerotics** A deep blue colour of the sclerotics is common in osteogenesis imperfecta (*fragilitas ossium*), a rare disease characterized by bony deformities and multiple fractures. It may also be seen in Marfan's syndrome and Ehler's Danlos syndrome. Dark grey or lead-coloured sclerotics may be noted in argyria after the oral use of silver salts for a long time. Pearly white sclerotics are common in idiopathic hypochromic anaemia.

## THE CORNEA

The transparent portion of the outer covering of the anterior aspect of the eyeball is called the cornea.

**Arcus senilis** *Arcus corneae senilis* is a fairly common degenerative condition of the cornea, characterized by an ill-defined greyish-white crescent or circle just within the outer margin of the cornea. It is lipid infiltration of the corneoscleral junction of the eyes and is considered to be a part of the natural process of aging. The ring is separated from the limbus by a small zone of clear iris. This clear zone distinguishes Kayser-Fleischer ring from arcus senilis. Unilateral arcus has been reported with contralateral carotid occlusive disease.

**Kayser-Fleischer ring** This is a characteristic golden brown pigment forming a complete ring (Fig 5.50) or at times only a crescent just internal to the



Fig 550 The Kayser-Fleischer ring



Fig 565 Bright red tongue of niacin deficiency



Fig 567 Cyanosed tongue



Fig 566 Atrophic glossitis

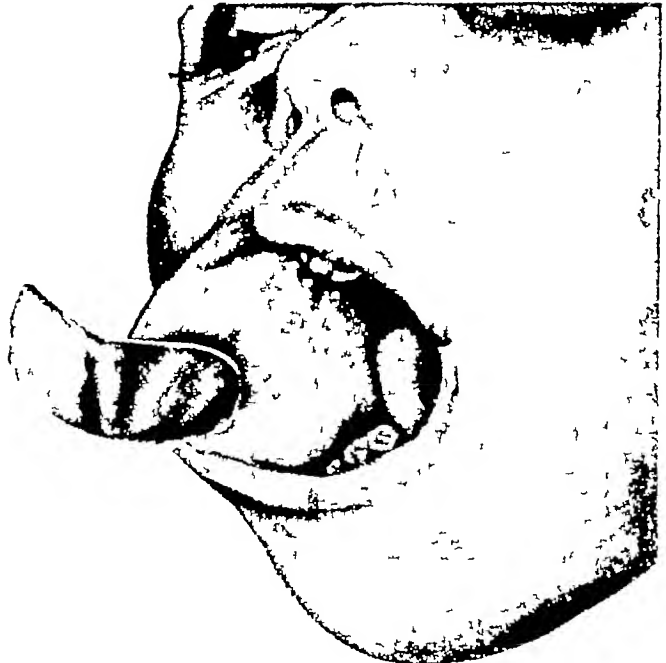


Fig. 576 Koplik's spots





limbus on the posterior surface of the cornea. Visible at times to the naked eye using oblique light, its presence should be confirmed by slit-lamp examination. It is diagnostic of hepato-lenticular degeneration, and occurs in no other condition.

**Interstitial keratitis.** Localized or diffuse dull grey areas of clouding or opacity over the cornea (due to cellular infiltration of the deeper layers of the cornea) in children or young adults, particularly when associated with Hutchinson's teeth and a "saddle nose", suggest congenital syphilis. The visual acuity may be markedly affected.

**Corneal opacities or opaque scars on the cornea** are usually indicative of old injuries, ulcers, inflammations or gonococcal ophthalmia.

**Hypopyon.** A collection of pus, in the anterior chamber of the eye, showing through the transparent cornea.

**Avitaminosis A.** Deficiency of vitamin A is associated with night blindness (nyctalopia), dry and shrunken conjunctiva (xerosis or xerophthalmia) and an opaque, cloudy or glassy cornea (keratomalacia).

**Ariboflavinosis.** In riboflavin deficiency, there is superficial vascularization of the cornea, with photophobia, lacrymation and angular cheilosis.

**Exposure keratitis.** In exophthalmos from any cause and in Bell's palsy, the exposed cornea may become dry and ulcerate (keratitis lagophthalmos).

**Phlyctenular keratitis.** Small greyish or yellowish nodules over the cornea or conjunctiva (phlyctenular conjunctivitis) may appear in debilitated children and last for days or weeks.

**Corneal ulcer.** A localized erosion or loss of substance of the cornea constitutes an "ulcer". Ulcers of the cornea are both important and varied. Traumatic, catarrhal, tuberculous, malignant, dendritic, hypopyon and Mooren's types of corneal ulcers are recognized. They may also occur in cases of Bell's palsy, exophthalmos or herpes zoster affecting the eye.

**Corneal calcification.** This is said to be the only true sign of hypercalcaemia. It is often difficult to distinguish from arcus senilis. It is usually a thin band, best seen on the medial side and is separated from the conjunctiva by a clear band of cornea.

## THE IRIS AND LENS

**Colour.** The colour of the eye mainly depends on that of the iris. The colour of the iris may be any shade of brown, grey, green, blue or even violet (as in some infants) or pink (as in albinos). The blueness of the iris in blonde subjects is due to absence of the normal brown pigment in the outer layers of the iris, the inner pigment shining through the muscle layer giving the blue colour. The pigment of the iris may be arranged like the spokes of a wheel.

(rayed or radiating type), as a band around the pupil (zonal type) or as multi-coloured specks and strands (mixed type) Shimmering irides have been described in Marfan's syndrome

**Iritis** Painful discoloration and swelling of the iris with circum-corneal vascularity, and a small and irregular pupil with a sluggish light reflex *Idiopathic* iritis is usually unilateral and relapsing, whilst *syphilitic* iritis is bilateral, non-relapsing and associated with the secondary stage of syphilis

**Ectopic lens** In Marfan's syndrome *ectopia lentis* may be obvious or detected by observing tremulousness of the iris on turning the head from side to side.

**Cataract** Degenerative alteration of the lens of the eye resulting in an opacity or loss of transparency is called cataract A cataract may be congenital, senile, diabetic, post-operative or occupational (as in X-ray and radium workers and glass-workers) It may be associated with myotonia dystrophica, Marfan's syndrome, homocystinuria, Down's syndrome, Friedreich's ataxia, Werner's syndrome, polychondritis, Laurence Moon-Biedel syndrome, or rubella syndrome According to site, it may be lamellar, central, anterior cortical, posterior cortical or total In cataract formation, the pupil frequently appears grey instead of black and symptoms like 'black spots before the eyes', "doubling or tripling of observed objects", and gradual impairment of vision make an appearance

**Refractive errors** The normal eye is emmetropic or with no error of refraction, the rays of light traversing the eye coming to a sharp focus on the retina In *myopia* (short-sightedness), the rays of light come to a focus before reaching the retina, the opposite condition is called *hypermetropia* (long sightedness) Defective focussing of light rays due to improper curvature of a refracting surface in the eyes is called *astigmatism* Unequal refraction in the two eyes is called *anisometria*

## PUPILS

A detailed description of the pupils will be found in the chapter on neurology

## THE TEMPLE

The temple, temporal region or side of the head, behind the eye, is of some importance in medicine

Fractures are both common and dangerous in this region because of the frequency of middle meningeal artery haemorrhage with resultant cerebral compression.

Palpation of the temporary artery should be carried out for evidence of arteriosclerotic thickening It also affords a simple and useful method of counting or studying the arterial pulse

**Temporal arteritis** This is a clinical entity of elderly subjects characterized by tender painful and nodular swellings of the temporal artery and associated with constitutional symptoms

**Tenderness of the scalp** This is a common symptom, encountered in cases of herpes zoster, scleroderma, temporal arteritis, trigeminal neuralgia, psychoneurosis and diseases of the eyes, ears, teeth paranasal sinuses meninges and bones of the skull

## THE EAR

## DEVELOPMENTAL ANOMALIES

Congenital malformations, such as the absence of the helix, anthelix or lobule, presence of vestigial or accessory auricle or Darwin's tubercle, unduly large, prominent or protruding ears (in Marfan's syndrome), or misplaced ears, are noted from time to time

**Gouty tophi** (Fig 5 54) Small hard nodules, visible over the helix and anthelix, arising from the cartilage of the ear and containing biurate crystals (exuded at times through ulceration of the skin overlying the nodules), are pathognomonic of gout

**Cauliflower ear** (Fig 5 52) A grossly deformed, thickened and flexible ear, common in wrestlers and pugilists, and resulting from repeated injuries to the ear

**Local lesions** Boils or furuncles, sebaceous cysts, chondromata (Fig 5 53), local haematomata, cirroid aneurysms (Fig 5 54), lupus and other skin diseases are all fairly common over the external ear

**Pulsation of ear lobes** Movements of the ear lobes may occur in severe aortic regurgitation, thyrotoxicosis, coarctation of aorta or severe tricuspid regurgitation with right sided failure

**Otalgia** (Ear ache or ear pain) Pain in or around the ear is an extremely common and annoying symptom, arising through causes within or outside the ear (reflex or referred otalgia) The important causes of ear pain are otitis media or middle ear disease, furuncle or boil, impacted wax or foreign body in the ear, injury, insect-bite, chilblain or frostbite (in cold weather), gout, dental conditions (e g unerupted wisdom tooth), diseases of the pharynx, larynx, nasopharynx or tongue, parotid gland involvement, temporomandibular joint lesions, herpes zoster, sphenopalatine neuralgia, trigeminal neuralgia, tabes dorsalis, angina pectoris and aneurysm of the innominate artery or aorta

**Ear tenderness** Tenderness over the ear may be significant Local tenderness over the tragus suggests a lesion in the external auditory meatus, over the mastoid process, mastoiditis, in front of or behind the ear, a pre-auricular or posterior auricular lymphadenitis, mumps or parotitis

**Ear discharge** A discharge from the ear may be serous, purulent (otorrhoea), foetid, pure blood or pure cerebrospinal fluid A *purulent* or seropurulent discharge is common in otitis media, furunculosis and other infections of the canal A *foetid* discharge occurs in necrosis of bone, impacted foreign body, otomycosis (fungus infection) and cholesteatoma *Blood* from the ear suggests trauma, malignancy or fractured skull *Cerebrospinal fluid* may be (rarely) discharged through the ear after a fracture of the base of the skull

Common affections of the external auditory meatus are (1) furunculosis or boils (with intense throbbing pain and tenderness), (2) impacted cerumen or wax (with heavy pain, vertigo, tinnitus or deafness), and (3) otomycosis or fungus in the ear (with a greenish semi-solid collection in the ear containing flakes and debris)

**Discoloration** A bluish or black discoloration of the external ear may be due to cyanosis frost-bite, haematoma, ochronosis or angiomatous tumors

*Deafness and Tinnitus* are discussed in Chapter 13

## THE NOSE

The nose consists of the bridge or upper third (with underlying bone), and the middle and lower thirds (with underlying cartilage), the lower third being the tip of the nose. Over the tip of the nose, the skin is rich in sebaceous glands, poor in blood supply and closely adherent to the underlying cartilage, these facts explain the painful nature of boils and other skin lesions over the nose, as well as the high incidence of malignant disease.

### CONGENITAL DEFORMITIES

The nose may be unduly long or short, bifurcated (Fig 5 55) or tubular (like a proboscis) and with a single median eye (cyclopia)

**Enlargement.** Enlargement of the nose, from thickening or hypertrophy of the tissues, occurs frequently in cases of acromegaly, myxoedema and rhinophyma. A gumma may rarely cause enlargement and deformity of the nose (Fig 5 56)

**Rhinophyma (Rhinoscleroma)** (Fig. 5 57) A massive or bulbous nose, with coarse, pitted and flushed skin near the tip, is common in elderly subjects with acne rosacea, it is a striking but unimportant condition, frequently portrayed in art and literature

**Saddle nose** Depression of the bridge of the nose, resulting in a characteristic deformity (saddle nose), is common in congenital syphilis (from gummatous necrosis and collapse of the nasal bones), but may also be encountered in congenital or infantile myxoedema (due to poor cartilaginous bone formation) or after trauma.

**Broad nose** Undue broadness or width of the nose may be due to hypertelorism, bony tumour, chronic sinus infection, myxoedema, cretinism or (rarely) Leishmaniasis (Fig 5 58)

**Beaked nose** In Werner's syndrome and in progeria the patients have beaked noses and premature graying

**Chronic or persistent redness of the nose or its tip** may be due to chronic alcoholism, chronic dyspepsia, acne rosacea, lupus erythematosus (Fig 5 59), cirrhosis of liver, mitral stenosis, polycythaemia or undue exposure to the elements. Acute or transitory redness of the nose may be due to a boil or furuncle of the nose, which, although apparently insignificant, may lead to serious complications like cavernous sinus thrombosis or meningitis

**Active alae nasi** Excessive working of the alae nasi with respiration, is common in pneumonia, but may also be seen in neurosis, cardiac failure or laryngeal obstruction, the nostrils open widely during inspiration

**Atresia or collapse of the alae nasi**, resulting in a long and narrow nose, with nasal obstruction, may be congenital or acquired from the healing of a tuberculous or syphilitic lesion

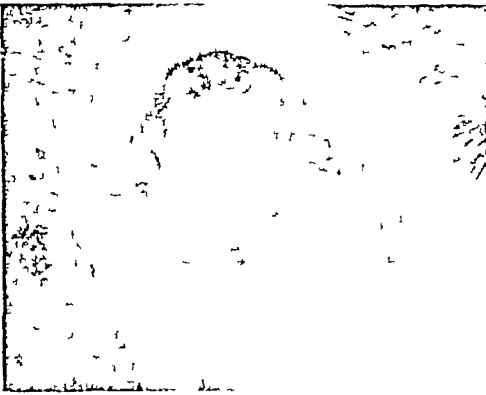


Fig 5.51 Gouty tophus of ear

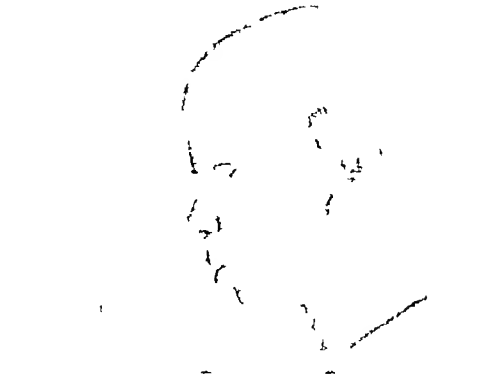


Fig 5.52 Cauliflower ear



Fig 5.53 Bilateral chondromata of ears

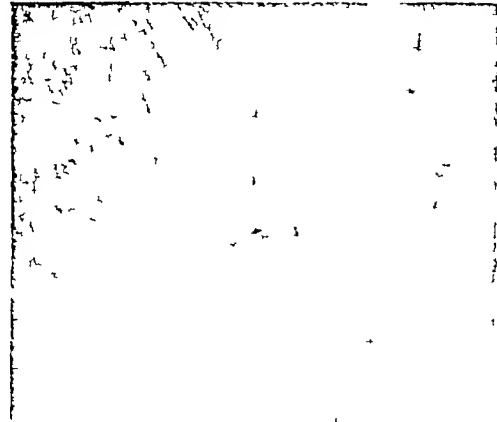


Fig 5.54 Cirroid aneurysm of ear

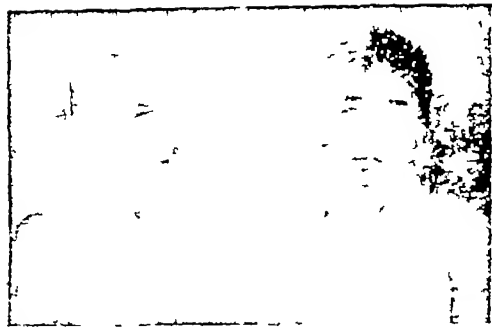


Fig. 5.55 Gumma of rose

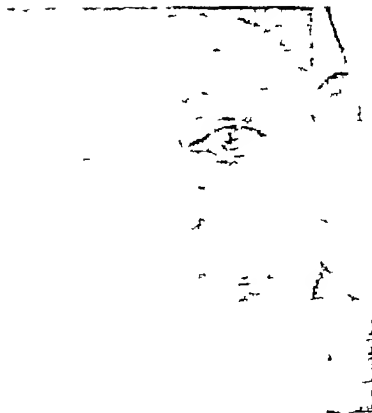


Fig. 5.56 Gumma of nose



Fig. 5.58 Leishmania tropica of nose

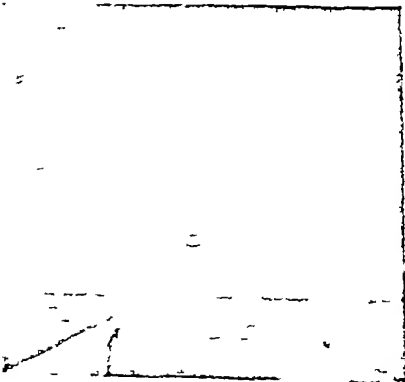


Fig. 5.59 Lupus erythematosus with butterfly patch on face.

**Nasal regurgitation of fluid on drinking**, suggests diphtheritic paralysis of the palate, bulbar palsy, cleft palate or perforated syphilitic palate

**Nasal pain** Severe pain in or around the nose may result from a boil or furuncle, ulcer, impacted foreign body in the nose, acute coryza, nasal diphtheria, sinusitis, Eustachian catarrh or engorged turbinate bone

**Sneezing** A sudden and violent expiration of air through the nose or sneeze, caused by direct or reflex stimulation of sensory nerves, is a common accompaniment of acute coryza, influenza, measles, hay fever or allergic rhinitis, vasomotor rhinitis, asthma and ear syringing. It may also be caused by drugs such as potassium iodide, snuff, pepper, fumes or exposure to sunlight

**Nasal discharge** A discharge from the nose may be watery, mucoid, muco-purulent, purulent, blood-stained, pure blood (epistaxis), foetid or foul discharge or pure spinal fluid (cerebrospinal rhinorrhoea). A *watery* discharge is usual in acute coryza, influenza, hay fever, measles, vasomotor rhinitis, trigeminal neuralgia, nasal polypi, diseases of the eye and after exposure to dusts, fumes or irritating substances

A *purulent* discharge occurs in the later stages of coryza or influenza, and in sinusitis, antral abscess and foreign body impaction

A *blood stained* discharge may be due to nasal diphtheria, impacted foreign body or nose-picking. *Epistaxis* (or "nose-bleed") may be caused by trauma, ulceration, polyp or new growth in the nose, impacted foreign body, hypertension, arteriosclerosis, rheumatic fever, typhoid, purpura and other haemorrhagic diseases (like haemophilia), scurvy, leukaemia, septicaemia, malaria, polycythaemia, high altitude, mountain-climbing, Osler's disease, or haemorrhagic hereditary telangiectasia, a rare heredo-familial disease with multiple angiomas and bleeding tendencies. *Foul* or *foetid* discharge from the nose suggests atrophic rhinitis (ozena), caries or necrosis of bone, syphilis, cancer, lupus or foreign body. Pure *cerebrospinal fluid* (watery fluid containing sugar and no albumin) oozing out of the nose suggests a fractured base of skull

**Rhinitis** An inflammatory condition of the mucous membrane of the nose may be of several types: acute catarrhal rhinitis (acute coryza or common cold), chronic rhinitis (hypertrophic or atrophic) or vasomotor rhinitis

**Nose-picking** A distressing habit, common in childhood, and usually due to scabs in the nose, nervousness or self-consciousness. It is seldom if ever due to intestinal worms or brain tumour, contrary to popular belief

**Nose obstruction** Due to congenital or acquired defects or diseases of the nose, nasal obstruction may lead to important side-effects, such as sniffing, noisy respiration, snoring, anosmia or loss of smell, mouth-breathing, deformity of face or chest, and alteration of voice

## THE CHEEKS

**Malar flush.** A pink or red flush on the cheeks may be due to mitral stenosis (mitral flush), acute form of pulmonary tuberculosis (hectic flush), lobar pneumonia, high fever, cold climate or high altitude (weather beaten appearance), menopausal syndrome (with transitory flushes), shy temperament, artificial beauty aids or a naturally "ruddy" complexion

**Visible vessels.** Spider angiomas and telangiectasis on the cheeks are common in liver cirrhosis



**Bell's palsy** or facial paralysis gives an asymmetrical appearance to the cheeks, the affected side showing a smoothed-out nasolabial furrow, a drooping or displaced angle of the mouth and a puffed-out cheek

**Involuntary movements** or spasms, involving the cheek or cheeks, are fairly common and may be of the nature of habit-spasm or tic, facial myoclonus (as in encephalitis lethargica), chorea, athetosis, clonic facial spasm or caused by herpes zoster of the geniculate ganglion

**Chvostek's sign** A light tap over the facial nerve, in front of the ear, in case of overt or latent tetany, results in a sudden contraction of the facial muscles

**Skin lesions** The skin of the cheek is prone to acne, pustular eruptions, boils, carbuncles, lupus erythematosus, lupus vulgaris, acne rosacea and adenoma sebaceum Septic lesions of the cheek are dangerously prone to intracranial complications in view of the anastomosis of the facial with the ophthalmic veins

**Hollowed cheeks** are a feature of serious illnesses, long-continued fevers and wasting diseases, and result from loss of fat from the so-called "suction pad", in front of the malar bone and masseter muscle

## THE LIPS

**Harelip** (Cleft lip) A congenital defect of the upper lip caused by lack of fusion of premaxillary and maxillary processes, during foetal life It is characterized by a deep cleft or indentation on one or both sides of the lip (unilateral or bilateral harelip) and at times associated with a cleft palate

**Cyanosis** Cyanosis is frequently detectable earlier over the lips than elsewhere, except in dark-skinned individuals and in those using cosmetics,

**Pallor** Pallor of the lips is frequently an early sign of anaemia and may be obvious earlier in the lips than elsewhere, provided this clue is not destroyed by the artificial application of "lipstick"

**Swelling** Marked swelling or thickness of one or both lips is suggestive of angioneurotic oedema (Fig 5 60), nephritis, nephrosis, traumatic biting of lip as in epilepsy, insect bite, abscess, carbuncle, cancrum oris or corrosive poison.

**Hypertrophy** of the skin and tissues of the lips may be racial (as in Negroes) or artificially induced (as in some African tribes) It is fairly common in acromegaly, myxoedema, cretinism and macrocheilia (massive hypertrophy of muscle and connective tissues of the lips)

**Thin lips** Thinness of the lips may be racial (as in American Indians) or familial

**Parched lips** Parched and dry lips are found in typhoid and other fevers, dehydration from cholera, dysentery, severe vomiting and diabetes mellitus

**Parted lips (Open mouth)** Lack of approximation of the lips is suggestive of obstruction in the nose or nasopharynx, large adenoids, dyspnoea associated with poor nasal airway, facio-scapulo-humeral myopathy, scleroderma, mongolism, cretinism, idiocy or imbecilism, bulbar palsy, hemiplegia, stomatitis, Parkinsonism or severe prostration

**Indrawn angle of the mouth** (or its deviation to one side) suggests facial paralysis, dental extraction or scarification from burns or cuts

**Twitching or tremors of the lips** may occur in old age, nervous exhaustion, hemiplegia, facial paralysis, bulbar palsy, epilepsy or general paralysis of the insane

**Foam or froth on the lips** may be noted in acute pulmonary oedema, epilepsy, hemiplegia, rabies or malingering

**Cheilitis** or inflammation of the lips may be due to allergy to cosmetics or toothpaste, excessive cold, infection or dental sepsis. Excessive peeling of the mucosa may keep on for months or years in bad cases (exfoliative cheilitis)

**Cracks or fissures** (angular cheilitis) at the commissures of the lips, with or without scaling, desquamation or bleeding, may be due to trauma (after dental extraction), local infection, stomatitis, anaemia, ariboflavinosis, diabetes mellitus, congenital syphilis, "*perleche*" or dermatitis (*pseudo-perleche*)

**Rhagades** Ulcerative fissures, at the angles of the mouth, in congenital syphilis, which often leave behind "linear, white, radiating scars", evidence of old, healed congenital syphilitic lesions (Fig 5 33)

**Perleche** Fissures or patches of wrinkling, maceration, inflammation or desquamation, at the angles of the mouth, and variously ascribed to vitamin B deficiency, low-grade streptococcal infection and other unknown factors

**Herpes labialis** (Herpes simplex, herpes febrilis, "Cold sores", fever blisters) (Fig 5 61) Clusters of small painful vesicles superimposed upon an erythematous base and with subsequent crust formation are common over the lips, nose and chin, in case of pneumonia, meningitis, severe cold or sunburn

**Ulcers of the lips** are common and of diverse types *benign ulcer*, *pyogenic ulcer*, *tuberculous ulcer*, *syphilitic ulcer* or *chancre* and *malignant* or *epitheliomatous ulcer* (firm or hard, indurated, deep seated and bleeding ulcer usually on the lower lip)

**Chancre** (Fig 5 62) A large, fairly indurated, elevated, circumscribed painless sore or lesion especially on the upper lip, associated with enlarged regional lymphnodes, followed by ulceration and blood-stained discharge, is good evidence of primary syphilitic infection

**Tumours** *Epidermoid carcinoma*, or *epithelioma*, once common in clay-pipe smokers, usually affects the lower lip in middle-aged or elderly males and results in a slow-growing, firm or hard, indurated, deep seated painless nodule or ulcer with elevated pearly margins and nodular regional lymphnode enlargement. Other varieties of tumours observed over the lips are *benign cysts* (small, firm, translucent, bluish, cystic swellings), *cutaneous horns* (elongated, hard, horn-like lesions with a tendency to malignancy), *haemangioma* (reddish or purple, compressible and reducible swelling with an impulse on crying or coughing), *lymphangioma* (compressible but not reducible swelling), and *pigmented mole* (potentially malignant and therefore dangerous)

**Abnormal smile** This may be distinctive enough to suggest a diagnosis. The *transverse smile* of myopathy, the *sneering smile* (myasthenic sneer) of myasthenia gravis, the *fixed smile* or *suppressed smile* of scleroderma and hepatolenticular degeneration, the *unilateral smile* of Bell's palsy, the *sardonic smile* of tetanus, excessive smiling in mania, smiling without cause in schizophrenia, and complete absence of smiling in spite of provocation in melancholia, and the unduly slow, sluggish and sustained smile of low amplitude in Parkinsonism are all characteristic in their own way

**Impaired mobility of the mouth or failure to open or close** it may be due to spasm or paralysis of the masticatory muscles, local pain or swelling or to ankylosis of the mandible. *Spasm* of the muscles of mastication (*trismus* or *lockjaw*) may be caused by tetanus, strychnine poisoning, hysteria, epilepsy, intracranial lesions or dental conditions. *Paralysis* of the masticatory muscles may result from organic or functional disease of the fifth nerve or its nucleus, pontine lesion, intracranial tumour or basal meningitis. *Local pain* or swelling, limiting mobility of the mouth, occurs in mumps, quinsy, Mikulicz's syndrome, trichiniasis and diseases of bone. *Ankylosis* of the mandible may be due to affections of the temporo-mandibular joint (true ankylosis) or periarticular affections (pseudo-ankylosis)

**Drooping of saliva** from the mouth may be noted in Parkinson's disease, after apoplexy or cerebrovascular accidents, in old age and in inflammatory conditions of the mouth

**Telangiectasis of lips** It is associated with hereditary haemorrhagic telangiectasia. It may also be seen in CRST syndrome (calcinosis, Raynaud's phenomenon, scleroderma, telangiectasis)

**Lip tie** Due to multiple frenulae may be seen in Ehler's Danlos syndrome

**Pursed lips** Some patients with severe chronic bronchitis or emphysema seem to breathe more comfortably if they purse their lips

**Fish mouth** Depression of the corners of the mouth in obesity associated with Cushing's syndrome

**Carp mouth** Rigidity and fixed facial expression with corners of the mouth drawn downwards due to facial spasm in tetany

**Tapir mouth.** In facio-scapulo-humeral dystrophy the lower lip protrudes in a characteristic fashion

## THE BREATH

The character of the breath may at times afford information of diagnostic value, e.g. in alcoholism, diabetes, acidosis, uraemia, oral sepsis, lung abscess, bronchiectasis, serious liver disease, atrophic rhinitis, where the breath may display a characteristic smell or odour

**Halitosis** or "foul breath" may be due to bad oral hygiene, stomatitis, atrophic rhinitis, follicular tonsillitis, sinusitis, bronchiectasis, lung abscess or lung gangrene, ingestion of substances like onion or garlic, or excessive fermentation in the stomach or gastric neoplasm. In a patient with pyloric obstruction, the breath is ordinarily offensive at time of eructation

**Alcoholic breath.** Although unmistakable, it should not induce a false sense of security and prevent further examination in a comatose or drowsy patient, in order to exclude important associated entities like head-injuries, concussion and cerebrovascular accidents

**Fruity odour.** The sweetish or fruitish odour of acetone, not unlike that of "new-mown hay" or "chloroform", should make one suspect diabetes mellitus, diabetic coma, starvation, acidosis or dehydration (acetone breath)

**Urinous or ammoniacal odour.** In uraemia, the breath frequently displays an ammoniacal or urinous odour (uraemic breath)

**A mousy or musty odour** of the breath (foetor hepaticus) has been described in cases of serious liver failure and cholaemia. It is a sweet, slightly faecal smell that has been compared to the odour of a freshly opened corpse or mouse

**Pungent odour.** In organo-phosphorous compound poisoning there is a characteristic pungent odour with often frothing at the mouth

**Gangrenous odour.** In gangrene of the lung, the breath may be foul suggesting decomposing matter

## THE TONGUE

From ancient times, the tongue (like the radial pulse) has been regarded as an invaluable clinical indicator of health and disease. The term *glossology* has been proposed to cover this limited field of investigation

Since considerable information of diagnostic value can be gathered from inspection of the tongue, a routine observation of this nature is absolutely essential in every case. The dorsum, undersurface, margins and tip of the tongue should all be systematically inspected by daylight or (failing that) with the aid of an electric torch. The colour, size, shape, coating, anomalies, surface, mobility and local lesions (including lumps, vesicles, ulcers and fissures) are all noted

The normal "greyish-red and stippled" appearance of the dorsum of the tongue depends on several factors. The greyish-white colour is due to the epithelial tufts covering the filiform papillae and the "fur" or coating of the tongue (consisting of epithelial debris, food particles and micro-organisms), the normal red or pink stippling of the tongue is due to the fungiform papillae.

**Developmental anomalies** Amongst the numerous congenital malformations of the tongue may be mentioned the following, viz. tongue-tie (unduly short fraenum linguae, a common abnormality), unduly long fraenum (rare), ankyloglossia (fixation of the tongue by lateral folds of mucosa), congenital macroglossia (massive tongue), microglossia (small tongue), hemihypertrophy, hemiatrophy, aglossia (complete absence of tongue), hemiglossia (absence of half of tongue), and bifid or trifid tongue.

### Congenital lingual defects

The *fissured tongue*, also called grooved or scrotal tongue (Fig 5 63), is characterized by deep depression or furrows, which run mostly in a longitudinal direction starting near the tip and disappearing gradually at the posterior third of the dorsum.

**Median rhomboid glossitis** This term is a misnomer as it is not an inflammatory process but a developmental lesion, resulting from failure of fusion of the lateral segments of the tongue. There is an oval or rhomboidal, red, slightly elevated area, about 1 cm in width and 2 or 3 cm long contrasting in colour with the rest of the dorsum of the tongue.

**Geographic tongue** (Fig 5 64) A characteristic type of abnormality with circular or annular red and flattened patches or areas (caused by atrophied filiform papillae) interspersed with zigzag lines (hypertrophied filiform papillae with piled-up epithelium), imparting the appearance of a geographical map to the tongue. Of obscure etiology and seen most often in children, it tends to recur at intervals or may persist unchanged throughout life.

**Colour** The tongue is normally greyish-red in colour. Any deviation of colour from normal must be carefully noted.

A *pale* or *white* tongue may be due to anaemia, leukoplakia, excessive furring (or coating), or irritant substances (e.g., mercury bichloride, carbolic acid or sulphuric acid). A *bright red* or *scarlet red* tongue (so-called "angry" or "fiery" tongue) may be due to niacin deficiency (Fig 5 65), atrophic glossitis of anaemia (Fig 5 66), acute glossitis, acute infectious fevers (such as scarlet fever), irritants, (e.g., sodium or potassium hydroxide) or foodstuffs (e.g., berries or red sweets). A *dark red* or *bluish red* tongue may be due to polycythaemia vera, cyanosis, riboflavin deficiency, antibiotics, atrophic or anaemic glossitis (during exacerbations—so-called "beefy tongue"). A *purple* tongue has been described in polycythaemia, a *magenta* tongue in ariboflavinosis, and chronic recovering-pellagrous tongue, and *orange-red* tongue in polyarteritis nodosa and after antibiotics, a *blue* tongue in cyanosis (Fig

5 65) or after sweets or local applications, a *yellow* tongue in jaundice (rarely), acute yellow atrophy (a yellowish brown fur) and irritants (nitric or hydrochloric acid), a *black tongue* (melanoglossia) from fungus infection (actinomycosis), antibiotics like penicillin, iron, bismuth, opium, tobacco or charcoal. A *brown* tongue may result from acute yellow atrophy, uraemia, liquorice or chocolate.

*Black hairy tongue* is a condition in which thick brownish or black furry patches made up of densely matted, hypertrophied filiform papillae, cover sometimes more than half of the dorsum of the tongue. This may result from the use of antibiotic troches or strong oxidizing mouth washes.

**Patchy pigmentation:** Local areas of discoloration on the tongue may be of diagnostic significance. In the tropics, it is not uncommon to see patches of pigmentation on the tongue (Fig 5 68). They are of no pathological significance. Thus, *dark brown*, *sepia black* or *purplish* patches on the tongue may be due to Addison's disease, Negro ancestry, old standing glossitis, and rarely to pernicious anaemia, syphilis, mercury or silver poisoning. *White* or *yellowish-white* patches on the tongue may be due to leukoplakia (on the dorsum) or xanthelasma (on the sides). *Bright red* or *scarlet red patches* (especially at the tip and margins) are suggestive of early glossitis or niacin deficiency. A *black patch* on the tongue with a "matted-hair appearance" is usually due to melanoglossia.

**Furred tongue (Coated tongue)** The "fur" on the tongue usually consists of epithelial cells and debris, food detritus and micro-organisms. *Excessive furring* of the tongue (a common finding) may be due to mouth breathing, excessive smoking, milk-drinking, gastritis or dyspepsia, constipation, alcoholism, defective salivation, fevers, acute exanthematous infections, or cirrhosis of the liver. *Unilateral furring* may be a feature of trigeminal neuralgia or unilateral paralysis of the tongue. *Central furring* (with the margins and tip of the tongue spared) has been considered a suggestive sign of typhoid fever. A dry and thick *brown fur* on the tongue may be noted in acute yellow atrophy or uraemia. A *black coating* may be due to melanoglossia. A complete *absence of furring* may be noticeable in the glossitis of vitamin B deficiency or anaemia.

**Dry tongue:** The tongue in health is moist. Obvious dryness of the tongue may be due to prolonged fever (as in typhoid), dehydration (as in severe diarrhoea or vomiting, dysentery or cholera), after haemorrhage, dyspnoea, mouth-breathing, coma, prostration, uraemia, belladonna or atropine effect, psychoneurosis, anxiety state.

**Enlarged tongue (Macroglossia, megaloglossia)** A large tongue may be due to congenital macroglossia (a rare anomaly), acromegaly, myxoedema, cretinism, Hurler's syndrome, angioneurotic oedema, acute inflammation or

abscess of tongue, insect bite, tumour, lipoma, haemangioma (Fig 5 69), lymphangioma, amyloid disease, cellulitis or advanced malignancy, or congenital hemihypertrophy (unilateral enlargement)

**Small tongue** (Microglossia, atrophy) An undue smallness of the tongue may be noted in dehydration (as in cholera or dysentery), haemorrhage, starvation or malnutrition, atrophic or anaemic glossitis (during remission), bilateral or unilateral paralysis (from hypoglossal involvement), typhus ("parrot tongue"), facial hemiatrophy (unilateral smallness of tongue), pseudobulbar palsy (small, pointed or conical tongue) and in Simmond's disease

In myasthenia gravis, atrophy of the tongue may be curiously selective, giving rise to triple longitudinal furrowing Although rare, this condition is pathognomonic of myasthenia gravis

**Tremor of tongue** A tremulous tongue may be noted in a variety of disorders, such as paralysis agitans, postencephalitic Parkinsonism, general paralysis of the insane, delirium tremens, bulbar palsy, involvement of hypoglossal nerve or nucleus, encephalitis, disseminated sclerosis, prolonged fevers, status typhosus, wasting diseases, cirrhosis of liver, neurosis, senility, chronic alcoholism, opium poisoning and excessive smoking

**Lizard tongue** (watch-spring or Jack-in-box tongue) After protrusion the tongue is shot back into the mouth at lightning speed and the lips are rapidly closed together It is seen in rheumatic chorea

**Spasms of tongue** These may be tonic (with the tongue becoming small, rigid and conical) as in nervousness, hysteria or general debility, or clonic spasms (the tongue displaying sudden jerks) as in chorea, epilepsy, habit spasm, G P I, disseminated sclerosis, hysteria or stuttering

**Deviated tongue** Deviation of the tongue to one side on protrusion may be due to unilateral paralysis from involvement of the hypoglossal nerve or its central connections (usual cause), malignant infiltration of the tongue, scarification after burns or severe ulceration or facial paralysis In nuclear or infranuclear hypoglossal lesions, the tongue appears unilaterally atrophied and displays fasciculations

**Immobile tongue** (Paretic tongue) A tongue that remains motionless on the floor of the mouth on attempt at protrusion suggests either bilateral lingual paralysis (bilateral involvement of the hypoglossal nerves or central connections), advanced malignancy of the tongue, bulbar palsy, amyotrophic lateral sclerosis, syringomyelia or tabes dorsalis

**Protruded tongue** In cases of mongolian idiocy, cretinism or macroglossia, part of the tongue remains permanently protruded outside the mouth

**Fissured tongue** Fissures on the tongue may be due to vitamin B complex deficiency, anaemias (atrophic glossitis), mongolian idiocy, acromegaly, "scrotal

tongue", acute glossitis, dental trauma, senility, bad oral hygiene, or congenital malformation

**Scarred tongue:** Scars on the tongue may be traumatic, secondary to ulcers from "tongue-biting" (as in epilepsy or bulbar palsy)

**Pellagrous tongue (Fig 5 65)** Deficiency of niacin (nicotinic acid and nicotinamide) besides causing hyperaesthesia or burning of the tongue results in any one of the following varieties of glossitis (depending on the degree and duration of the deficiency), viz. (1) *acute apico-marginal* or *early glossitis* (with a characteristic redness of the tip and margins of the tongue), (2) *acute diffuse glossitis* (the so-called scarlet-red, fiery or angry tongue), (3) *ulcerative* or *membranous form* (due to secondary infection, with formation of small ulcers and gray membranes on a red base), (4) *Moeller's glossitis*, a localized and migrating form of glossitis (the so-called bald, smooth, polished or varnished tongue, devoid of papillae and either red or pale in appearance)

**Ariboflavinosis tongue:** In riboflavin deficiency, involvement of the angles of the mouth (cheilosis or perleche), buccal mucosa and conjunctivae are earlier and more important than affection of the tongue. When affected the tongue displays (1) a characteristic magenta or dark red colour and (2) a "cobblestone" appearance (due to hyperaemic and hypertrophied papillae covered with thickened epithelium)

**Atrophic glossitis (Smooth or bald tongue) (Fig 5 66)** Atrophy of the papillae resulting in a smooth (polished or varnished) or glossy tongue may result from a variety of disorders: megaloblastic anaemias (e.g., pernicious anaemia, nutritional megaloblastic anaemia, tropical sprue, pregnancy anaemia and pancreatic steatorrhoea), iron deficiency anaemia (microcytic hypochromic anaemia), chronic vitamin B complex deficiency, gastritis with achlorhydria, malignancy and through unknown causes (primary glossal atrophy)

Two main types of appearances are presented. (1) During active phases (or exacerbations) of the causative anaemia or deficiency, the tongue is diffusely swollen, painful, raw, angry and bluish-red, dusky red, or the colour of beef-steak (beefy tongue). On its surface may be seen red puncta, minute ulcers or erosions and haemorrhagic spots. (2) During quiescent phases or remissions the tongue appears small, smooth, shiny, polished or varnished, furrowed or ridged and faint pink, salmon pink or dusky red in colour.

**Raspberry tongue.** Scattered red dots on a grey background give a characteristic raspberry appearance to the tongue in the early phases of scarlet fever. It is due to red fungiform papillae sparsely dotted over the grey tongue.

**Strawberry tongue.** In the latter phases of scarlet fever, with further progression of epithelial exfoliation, all the papillae (fungiform and filiform) appear as large red knobs on the tongue giving it the so-called strawberry appearance.

**Cobblestone tongue.** A cobblestone appearance of the tongue has been described in ariboflavinosis (with a magenta colour), syphilis (with leukoplakia) and after antibiotics (antibiotic tongue).



**Smoker's patch** A small, raised, smooth, congested area, frequently covered with a crust on the front of the dorsum of the tongue

**Syphilitic glossitis** The nature of the lesion is mainly dependent on the stage of the syphilitic infection. The main types of lesions are

(1) *Primary chancre* (Fig 5 70) A small, superficial, hard, indolent, oedematous nodule, sore or ulcer near the tip in males, associated with sub-maxillary and submental lymphadenopathy in the primary stage

(2) *Hutchinson's wart* A small, hard, wart or condyloma on the dorsum, occasionally encountered in the secondary stage

(3) *Mucous patches* and *snail-track ulcers* Multiple, small, shallow ulcers over the tongue and buccal mucosa with shotty lymphnodes in the secondary stage of syphilis

(4) *Localized gummata* One or more globular, firm, painless, progressive swellings, superficially or deeply embedded in the tongue, in the tertiary stage. They may lead to gummatous ulcers

(5) *Leukoplakia* (Leukokeratosis, ichthyosis linguae), hypertrophy and cornification of the epithelial surface leading to the formation of thick, white or opaque, square, round or oblong raised plaques, giving a white appearance to a part of the tongue (Fig 5 71). Leukoplakia may be encountered in tertiary syphilis and may be regarded as a potentially pre-cancerous condition

(6) *Glass tongue* (Sclerosing glossitis) A smooth, polished, small and hard tongue, found occasionally in cases of tertiary syphilis and distinguishable from the tongue of atrophic glossitis by its hardness on palpation. It is due to atrophy secondary to syphilitic endarteritis obliterans

**Amyloid tongue** An amyloid tongue is usually a part of a generalized amyloidosis. Only occasionally are isolated amyloid deposits found in the base of the tongue. The tongue is enlarged and presents a mottling of dark purple areas with translucent matter

**Ulcers** Ulcers of the tongue, of which there are many varieties, may be single or multiple. A *single ulcer* may be carcinomatous (epitheliomatous) (Fig 5 72), tuberculous, syphilitic, dental or fraenal. *Multiple ulcers* of the tongue may be due to dyspepsia (dyspeptic ulcers) or ulcerative stomatitis, including aphthous stomatitis and Vincent's disease, secondary syphilis, herpes, pemphigus, small-pox, chicken-pox, eczema and vitamin B-complex deficiency

A *carcinomatous ulcer* of the tongue is usually single, more often on the side or tip of the tongue, very hard and indurated, irregular, deep, associated with a slough, impaired mobility of the tongue, regional lymphnode enlargement and with raised everted margins, usually in elderly males and very resistant to treatment. Non-ulcerative forms of tongue carcinoma are the scirrhus, nodular, papilliferous and fissured forms

A *tuberculous ulcer* is rare, usually at or near the tip, painful, not hard, small, with a granulated base and thin undermined edges, with evidence of tuberculosis elsewhere and usually in young individuals

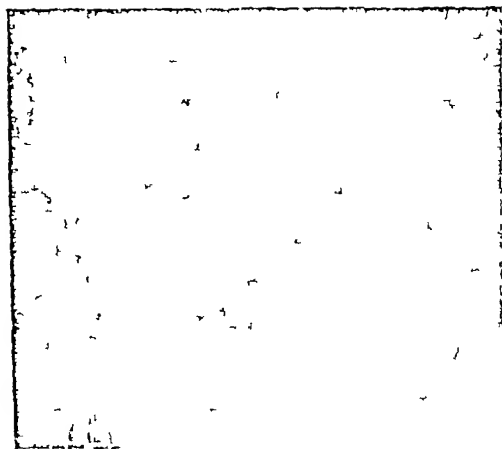


Fig 560 Angioneurotic oedema of tongue and lips

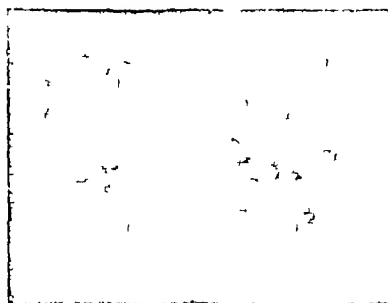


Fig 561 Herpes labialis

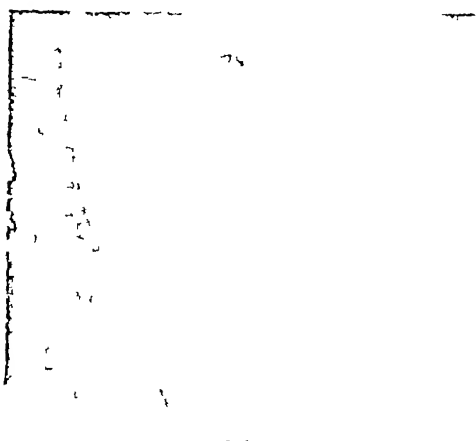


Fig 562 Chancre of lower lip



Fig 563 Scrotal tongue

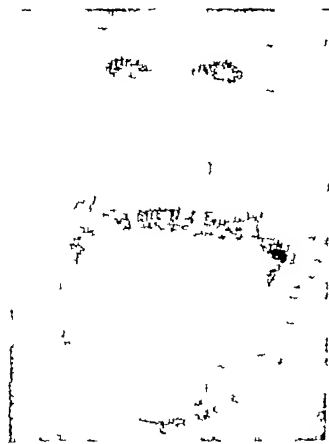


Fig 564 Geographical tongue



Fig 568 Pigmentation on tongue

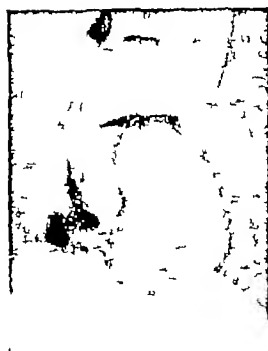


Fig 569 Haemangioma of tongue

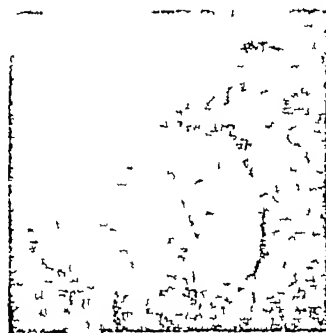


Fig 570 Primary chancre of tongue

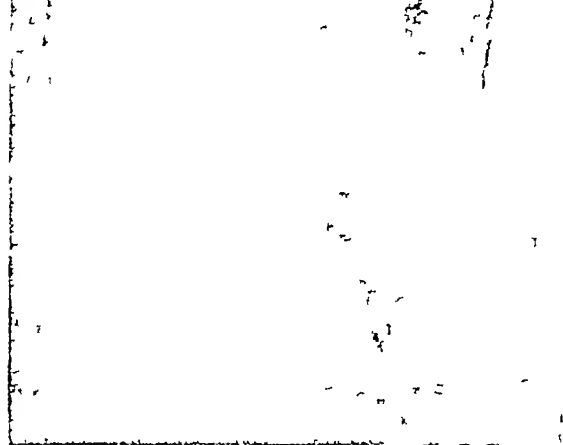


Fig 572 Early carcinomatous ulcer of tongue

leukemia tongue

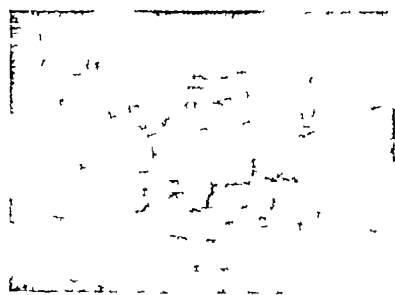
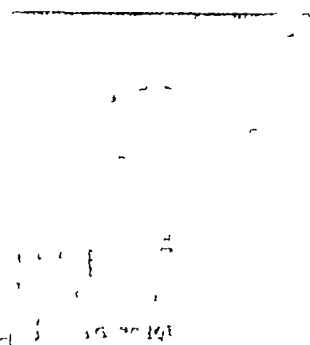


Fig 574 Bleeding gums in acute leukaemia

3 Scurvitic spongy gum

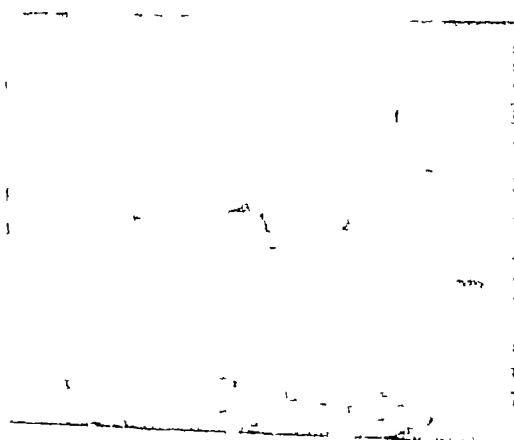


Fig. 575 Gross gingival hyperplasia owing to phenytoin sodium intoxication

A *syphilitic ulcer* may be single (as with primary chancre or solitary gumma of the tongue) or multiple as in secondary syphilis, in the latter condition, the ulcers (or mucous patches) are superficial, scattered all over the buccal mucosa, painful, small, round, with greyish bases and sharply punched-out appearances and associated with small and shotty lymphnodes

A *dental ulcer* is usually located opposite a jagged or broken tooth, on the margin of the tongue. It is small, superficial and non-indurated. A *fraenal ulcer* occurring on the fraenum of the tongue may be due to severe bouts of coughing, as in pertussis

**Parrot tongue** The small, shrivelled, dry tongue with a brownish fur of uraemia is at times referred to as the "parrot tongue"

**Truncated tongue** Rarely a tuberculous ulcer at the tip of the tongue associated with oedema of the rest of the tongue may impart an "amputated look" to the tongue

**Alligator tongue** A dry, thick, furrowed and irregular tongue in diabetes mellitus

**Caviar lesion** Varicosities of the sublingual veins may be seen on the under-surface of the tongue in elderly patients. The round shape and black colour of this cluster of vessels resembles caviar

**Fatiguability of tongue** In myasthenia gravis weakness of the muscles of the tongue leads to difficulty in articulation

## THE ORAL CAVITY

Examination of the oral cavity or mouth should be carried out, after removal of artificial dentures, systematically and with care, either in daylight (preferable) or with the aid of an electric torch or reflecting head-mirror. The tongue, teeth, gums, buccal mucous membrane, palate, pharynx, tonsillar regions should all be subjected to a visual examination, preferably with the aid of a tongue depressor

Inspection is then followed by palpation with the gloved finger or fingers (digital examination) to determine features like tenderness, size, shape, induration, consistency, texture, etc. of local lesions, particularly tumours and ulcers

## THE TEETH

The appearance of the teeth occurs in two stages and with two sets of teeth, viz. the temporary or deciduous teeth (20 in number) and the permanent teeth (usually 32 in number). The eruption time and order of appearance of the two sets of teeth are usually well-defined, particularly in the case of deciduous teeth

Dental ailments are frequently responsible for or predispose to diseases in other parts of the body, such as gastric disorders, arthritis, maxillary sinusitis and bacterial endocarditis

**Developmental anomalies (anomalous dentition)** may be noted in the form of anodontia (absence of all teeth), supernumerary teeth (e g , palatal teeth), extra or fourth molars (behind the wisdom teeth) or a third set of teeth (third dentition in old age)

**Early (or premature) dentition** A premature eruption of teeth is of no pathological significance

**Delayed (or retarded) dentition** This may be due to rickets, cretinism, juvenile myxoedema, mongolism, mal-nutrition or vitamin deficiencies

**Difficult dentition** may be due to anomalous position or impaction of an erupting tooth or to unduly small or narrow jaw-bone formation. Mal-position (faulty or irregular arrangement of teeth) may result from premature loss of deciduous teeth or retarded appearance of permanent teeth

**Impacted tooth** An erupting or unerupted tooth, such as the wisdom tooth, may get obliquely wedged against an adjacent tooth resulting in considerable pain and swelling

**Hutchinsonian teeth (Hutchinson's teeth, notched teeth)** Pathognomonic of congenital syphilis, the upper central permanent incisors are rounded or peg-like, widely spaced or separated, show a characteristic crescentic notch on the biting edge and taper towards the free margin. In conjunction with interstitial keratitis and deafness, Hutchinson's teeth complete the diagnostic "triad" of congenital syphilis

**Moon molars ("Mulberry molars")** The first molars of the second dentition erupt at about the sixth year and present a deficient development of the cusps with hypertrophy of the surrounding enamel. They are called moon molars because they are dome-shaped due to tapering of the crown of the tooth. They have been also called mulberry molars because the cusps are stunted and rounded

**Wide spacing of the teeth** may be due to acromegaly, von Recklinghausen's disease (hyperparathyroidism) or extraction of adjacent teeth

**Serrated (or dentated) teeth**, with serrated biting edges, are a sign of mal-nutrition

**Pitted teeth** may be due to fluorosis or excess of fluorine in the drinking water

**Furrowed (or striated) teeth** Transverse grooves or striae on the teeth are indicative of serious diseases or fevers in childhood, rickets and hypoparathyroidism

**Mottled enamel** In fluorosis or with excess fluorine in the water supply, there is yellowish-brown mottling of the teeth.

Loose (or mobile) teeth may be due to old age, scurvy, pyorrhoea alveolaris, trauma, hyperparathyroidism, severe stomatitis, mercury or phosphorus poisoning or purpura haemorrhagica

**Projecting teeth** may be congenital, familial or due to acromegaly

**Attrition** or shrinking of the teeth, to almost total disappearance at times, may be due to the use of corrosive substances or constant biting of hard objects (e.g. betel-nut, cigarette-holder or pipestem)

**Teeth grinding** (or gritting), common in childhood, particularly during sleep, is usually due to nervousness, lack of sleep or improper digestion. It may be associated with rickets or intestinal helminthiasis

**Dental caries** Caries or decay of teeth may manifest itself as roughening, discoloration (milky white, yellow, brown or black areas), cavitation or destruction of the affected teeth. Early or extensive dental caries may be due to rickets, diabetes mellitus, pregnancy, osteomalacia, malnutrition, oral sepsis or to overproduction of bacterial acids in the mouth (probably from excessive carbohydrate intake)

**Pink teeth** giving a strong red fluorescence to ultraviolet light in a dark room, are considered diagnostic of the rare condition of porphyria

**Apical abscess** (or infection) This has been incriminated as a source of infection (septic focus) in a variety of ailments, frequently without justification. The condition can only be detected by radiological examination

**Tumours** arising in association with the teeth are (1) dentigerous cysts, (2) odontomas (tooth-like structures) and adamantinomas (epithelial tumours). Diagnosis of these tumours is usually suggested by an expansion or thickening of the jaw bone and verified by roentgenological examination

## THE GUMS (GINGIVAE)

**Pallor** Pallor of the normally pink gums may be due to anaemia or the various other causes of skin pallor

**Cyanosis** Generalized cyanosis from any cause may be strikingly manifested in the gums as a deep blue or bluish coloration

**Bright red colour** of the gums suggests inflammation or gingivitis

**Discoloration** or pigmentation of gums, usually patchy or linear, may be due to poisoning by lead, bismuth, mercury, silver or other heavy metals. In lead poisoning, a characteristic lead line on the gums (usually adjacent to unhealthy teeth) may clinch the diagnosis

**Spongy, swollen red or bluish-red and sore gums**, with a tendency to bleeding, are common in scurvy ("scurbutic gums") (Fig 573), but may also be noted in purpura, leukaemia, (Fig 574), vitamin deficiencies, diabetes mellitus, metallic poisoning (e.g., mercury poisoning), faulty oral hygiene, agranulocytic angina and aplastic anaemia

Atrophy or shrinking of the gums may follow acute gingivitis, or Vincent's angina and may arise in wasting diseases or senility *Hypertrophy* of the gums is common after the drug treatment of epilepsy, in endocrine disorders and in pregnancy

Necrosis or sloughing of the gums may arise occasionally in cases of agranulocytosis (especially after the use of certain drugs), vitamin deficiencies, malnutrition, severe gingivitis, Vincent's angina and at times after dental extractions

*Pyorrhoea alveolaris* A common disease involving teeth and gums, with formation of pus-containing "pockets" between the teeth and gums, later leading to the loosening of teeth, retraction of gums, destruction of bone and formation of gingival abscesses

Necrotizing ulcerative gingivitis (Vincent's angina), See ulcero-membranous stomatitis

Tumours (Epulis) A benign tumour arising from the gum or peridental structures is frequently called an "epulis" Several varieties of tumours, both benign and malignant, are recognizable, viz., (1) fibroma (a slow-growing fibrous tumour with a normal mucosal covering), (2) giant cell tumour (a soft, nodular or smooth, localized tumour), (3) "brown tumours" of hyperparathyroidism, (4) inflammatory or granulomatous epulis or tumour, (5) sarcoma, and (6) carcinoma.

Dilantin gums Gingival hyperplasia develops in a fairly large percentage of patients receiving the anti-convulsive drug dilantin (Fig 575) It occurs especially in children and young adults Usually, the process is painless Edentulous portions of the gums do not manifest hyperplasia.

## THE BUCCAL MUCOSA

The buccal mucous membrane should be carefully inspected with the aid of a tongue depressor, as it may display a variety of significant lesions or signs of disease

**Buccal pigmentation** Pale brown or grey patches or streaks of pigmentation on the inner aspects of the cheeks and over the palate, gums or tongue should be carefully noted, being of diagnostic significance In the great majority of cases, they are diagnostic of Addison's disease (especially when associated with generalized skin pigmentation, low blood pressure, vomiting and asthenia) Occasionally, buccal pigmentation is due to Negro ancestry, pernicious anaemia, poisoning with arsenic, bismuth, silver or lead, pulmonary tuberculosis, syphilis or malignancy

Fiery redness or a scarlet-red appearance of the buccal mucosa (in association with a similar appearance of the tongue —so-called "angry" or "fiery" tongue) suggests niacin deficiency In riboflavin deficiency, the mucosa has a diffusely dull-red colour

**Fordyce's spots** Small, usually discrete and scanty, yellowish-brown, slightly raised spots may be a normal finding, over the mucosa of the cheeks and lips, in many persons

**Koplik's spots** (Fig 5 76) Small bluish-white spots on an erythematous base are frequently observed on the buccal mucosa, opposite the molar teeth in the early or pre-eruptive stage of measles. They frequently afford an early clue to diagnosis

**Exanthematous lesions** During the eruptive stage of exanthematous fevers like measles, small-pox, and chicken-pox, eruptions are commonly encountered over the buccal mucosa.

**Herpetic lesions** Painful vesicular lesions in clusters over the erythematous base (or minute ulcers in groups) over the buccal mucosa may be due to herpes simplex (herpetic gingivostomatitis) or herpes zoster

**Stomatitis.** Stomatitis means inflammation of the buccal mucosa and may be due to a variety of factors. The mucosa appears red, swollen and tender with excessive salivation and sometimes tendency to bleed. The common clinical types are (1) *Catarrhal stomatitis*. The mucosa is red and there is increased exudation from the mucous glands. It may be due to local causes such as poor oral hygiene, or use of broad spectrum antibiotics, or result from general debility or infectious disease. (2) *Aphthous stomatitis* (canker sores), may affect lips, cheeks or tongue. It starts as one or more vesicles which rupture leaving painful ulcers which heal spontaneously. (3) *Ulceromembranous stomatitis* (Vincent's angina). The ulcer surface is covered by a greyish pseudo-membranous slough demarcated from the surrounding mucosa by a linear erythema. Associated features are increased salivation, foetid odour, spontaneous gingival bleeding, lymphadenopathy and fever. (4) *Thrush*, due to fungus infection occurs in infants, debilitated adults such as heroin addicts, and in patients treated with corticosteroids and antibiotics. It presents as whitish patches which are easily removed leaving a raw bleeding surface.

**Leukoplakia** The white translucent plaques of leukoplakia, although commoner over the tongue, may be noted over the buccal mucosa, in cases of long-standing atrophic glossitis, syphilis or excessive use of tobacco.

**Petechial haemorrhages, purpuric spots or ecchymoses** may be noted in the mucous membrane of the mouth in cases of purpura, haemorrhagic diseases, subacute bacterial endocarditis or septicaemia and disseminated lupus erythematosus. Their appearance in the mouth should be viewed with caution as they frequently precede haemorrhagic phenomena.

**Mucous patches (Snail-track ulcers)** In secondary syphilis, the mucosa of the buccal cavity, tongue and lips may show characteristic painless, slightly elevated, circumscribed, round or oval lesions or ulcers covered by pearly-white or grey membrane, the patches tend to bleed on scraping and show regional lymphnode enlargement.



**Cancrum oris** (Noma, gangrenous stomatitis) A rare form of stomatitis in debilitated children after measles and other infections, characterized by extensive ulceration, sloughing and necrosis, involving the cheek and gums and leading to death from exhaustion

## THE PALATE

The anterior three-fourths of the roof of the mouth or palate is formed by the hard palate and the posterior one-fourth by the soft palate

**Cleft palate** A median, symmetrical defect or perforation of congenital origin in the hard palate, caused by lack of fusion of the embryonal palatal processes. It may lead to regurgitation of food or drink through the nose. It is frequently associated with congenital hare-lip.

**Torus palatinus** A median ridge or linear elevation of the hard palate at the site of fusion of the embryonal maxillary processes. It is a developmental anomaly of no significance.

**High arched palate** (Gothic palate) A high palatal arch may be a harmless developmental anomaly or commonly occurs in congenital heart disease. It may also be seen with Marfan's syndrome, trisomy 18 syndrome, Turner's syndrome, Pteris Robin syndrome (hypoplastic mandible with shrew-like face), etc. or due to nasal obstruction in childhood (from adenoids or deviated septum), or associated with malposition of teeth or arachnodactyly.

**Perforation of palate**, resulting at times in the regurgitation of fluids through the nose, may be due to a congenital cleft palate, syphilis, tuberculosis, actinomycosis, diphtheria or trauma.

**Anaesthesia of palate** Loss of sensation over the palate may be encountered in hysteria or paralysis of the trigeminal nerve (sensory portion).

**Paralysis of soft palate** Either unilateral or bilateral paralysis or paresis of the soft palate may be due to bulbar or pseudo-bulbar palsy, post-diphtheritic paralysis or to operation or trauma. In unilateral paralysis, one side alone of the palate moves upwards on articulating the sound "ah", in bilateral paralysis, there is no upward movement at all of the palate, the voice has a nasal twang and regurgitation of fluid occurs through the nose.

**Vesicles** Vesicles on the palate may be due to herpes zoster, herpes simplex, exanthematous fevers such as small-pox or chicken-pox, or skin diseases.

**Ulcers on the palate** may be due to herpes, various forms of stomatitis, syphilis, tuberculosis, malignancy, deficiency diseases or blood dyscrasias.

**Tumours** A tumour of the palate, when hard and smooth, suggests either an exostosis or echondroma, when soft, nodular and rapidly growing, either a malignant tumour or a myxoma.

## FLOOR OF THE MOUTH

The following structures are normally distinguishable, on the floor of the mouth, with the tongue upturned, viz the fraenum linguae (a fold of mucosa

connecting the undersurface of the tongue to the floor of the mouth), the sublingual ridge (corresponding to the sublingual gland), and Wharton's papilla (a papilla which marks the opening of Wharton's duct and at times of the duct or ducts of Bartholin)

**Ranula.** A cyst under the tongue on the floor of the mouth, usually called a ranula, may be a retention cyst of a mucous gland, an obstructive cyst of a salivary gland or a congenital inclusion cyst. A true dermoid cyst is not included under this term.

#### FAUCIAL TONSILS

The faucial tonsils are oval structures occupying the tonsillar fossae. On each tonsillar surface are crypts (or recesses) leading into follicles (or pockets).

**Acute tonsillitis.** This may be primary (and due to one or more of a large number of organisms) or secondary to some acute exanthem like scarlet fever or measles. Acute tonsillitis may display several distinct forms, viz. acute catarrhal form (the surface being flushed and inflamed), acute parenchymatous form (the whole tonsil being engorged and swollen), acute follicular form (with involvement mainly of the follicles) and the benign membranous form (with a dirty yellow or grey membrane, easy to remove and without bleeding). Rarely, a tonsillar abscess may supervene on an attack of acute tonsillitis.

**Chronic tonsillitis:** The enlarged tonsils of chronic tonsillitis owe their size to parenchymatous hyperplasia, fibroid degeneration or distention of follicles. Enlargement of tonsils *per se* is not considered significant unless accompanied by exudation of pus, severe congestion, regional lymphnode enlargement or repeated attacks of acute inflammation.

**Peritonsillar abscess (Quinsy).** A localized collection of pus adjacent to or outside the tonsil proper is called "quinsy". Its main characteristics are high fever with rigors, severe pain in the jaw, ear and neck on one side, local tenderness, lymphadenitis, bulging of the anterior pillar and soft palate on one side, impaired mobility of the soft palate unilaterally and a local layer of pus.

**Diphtheria.** A serious condition of the throat distinguished by the formation of a characteristic grey or yellowish white, adherent and spreading membrane with a tendency to bleed on scraping, covering the tonsils, fauces and pharyngeal wall and accompanied by enlarged and tender lymphnodes. The accompanying fever and pain may be less obvious than with acute tonsillitis but the prostration is much greater.

**Infectious mononucleosis.** Here the membrane has a striking white colour and is confined to the tonsils.

**Agranulocytosis.** Initially there is swelling and redness of the throat followed by the formation of a yellowish or greyish-black membrane which sloughs to produce ulceration.

# 6 | The Neck

## EXAMINATION OF THE NECK

THIS must always be carried out in a good light, with the patient sitting and facing the examiner, and in the following order inspection, palpation and auscultation

**Inspection** The neck is carefully surveyed for the following features, viz general appearance, position, deformities, asymmetry, limitation of movements, abnormal movements, size and shape of the thyroid gland, presence of other swellings, and engorgement or pulsations of blood vessels After surveying the neck from the front, the head is slightly extended and rotated to each side in turn, in order to define the sternomastoid muscle and the anterior and posterior triangles on either side The neck is then re-inspected from each side, particularly for lymphnode enlargements and other swellings and for pulsations

**Palpation** This is carried out systematically (preferably from the front, side and back of the patient), all normal and abnormal structures in the neck being palpated individually and preferably in the following order, viz, the lymphnodes, salivary glands, thyroid gland, trachea, blood vessels and all other swellings or abnormalities of the neck, superficial or deep Note is also made of active and passive movements of the neck, stiffness or wasting of neck muscles, areas of local tenderness or heat, and deformities of spine

**Auscultation** This may be practised routinely over the thyroid gland and blood vessels for evidence of murmurs or vascular sounds

## GENERAL APPEARANCE OF NECK

**Shape and size** A *thin* and *long* neck ("scraggy neck") is common with a hyposthenic constitution, in states of wasting, emaciation or cachexia, bronchial asthma, pulmonary tuberculosis, Simmond's disease, Addison's disease and anorexia nervosa

A *broad* and *short* neck ("bull neck") is common with the hypersthenic or plethoric habitus, in obesity (particularly of the "central", "superior" or "buffalo" type, as in Cushing's syndrome), cervical lipomatosis, overdeveloped neck muscles, cellulitis of neck, Ludwig's angina, myxoedema and cretinism. *Webbing* of the neck (Fig 6 1) is seen in the syndrome of ovarian dysgenesis, short stature and infantile development of accessory sex structures including the external genitals (Turner's syndrome) and in Klippel Feil syndrome.

**Cervical rigidity** ("Stiffness of neck") This results in lack of mobility, inability to flex or rotate the head and palpable hardness of the neck muscles. It may be due to a variety of causes, e.g., meningeal irritation (as in meningism, meningitis, encephalitis and subarachnoid haemorrhage), tetanus, affections of the cervical vertebrae (e.g., arthritis, caries or fracture-dislocation), infections of the neck, ear or mastoid, "stiff-neck" (from chill or strain), chronic torticollis and hysteria.

**Weak neck muscles** Undue weakness of the muscles may prevent extension and even the normal upright position of the head. This may occur in myotonic dystrophy, polymyositis, anterior horn cell disease, poliomyelitis, myasthenia gravis or emaciation.

**Torticollis** (Wry neck) (Fig 6 2) An abnormal deviation with rotation of the head to one side. Several varieties of torticollis may be encountered: acute rheumatic type due to myofibrositis of sternomastoid and trapezius muscles from chill or trauma, chronic or congenital type due to organic shrinking from birth of the sternomastoid muscle or defective development of the atlas or occipital bone, reflex type secondary to septic glands or cervical caries, hysterical type and a spasmodic type.

**Abnormal movements** These may be in the nature of a spasmodic torticollis (clonic jerking of the head with rotation) or of head nodding (nodding spasm, "salaam" spasm), the latter may be due to habit spasm, hysteria, chorea, epilepsy, old age, Parkinsonism, encephalitis or aortic regurgitation (Mussel's sign). Sudden, jerky, inconstant and variable movements of the head and neck are not unusual in chorea and hysteria.

**Absence or atrophy of sternomastoids** This may be quite noticeable on turning the head to one or other side or by forward flexion of the head against resistance. Complete absence of one or both muscles suggests a congenital anomaly, paralysis of the muscle means involvement of the spinal accessory nerve. Atrophy of the sternomastoids is invariably present in dystrophia myotonica and polymyositis.

**Head retraction** Hyperextension of the neck, from excessive rigidity or spasm of the neck muscles, as in meningitis, meningism, subarachnoid haemorrhage, encephalitis or tetanus. It may be part of an opisthotonus or generalized arching of the body and spine.

**Cervical oedema** Subcutaneous oedema of the neck may be due to generalized anasarca, superior vena caval syndrome, thrombosis of veins or inflammation of the mouth, neck or throat.

**Lipomatosis of neck** Lipomata or fatty tumours are common in the neck region, particularly at the back. *Cervical lipomatosis* or multiple lipomatosis of neck (with a "double" or "triple" chin) is not uncommon in chronic alcoholics and beer drinkers.

**Pachydermatocele** (*Elephantiasis neuromatosa*) Massive hypertrophy of skin and subcutaneous tissues (associated with neurofibromatosis) is particularly common over the neck (Fig. 6 3)

**Ludwig's angina** An inflammatory swelling with oedema of the submaxillary region and floor of the mouth causing protrusion of the tongue. Frequently secondary to oral sepsis, it may rarely cause death from oedema of the glottis.

**Cellulitis of neck** (Fig. 6 4) A serious condition, secondary to sepsis in the mouth or throat or diphtheria, associated with a rapid, tender and painful, red and brawny swelling of the neck and pressure symptoms.

## THE LYMPHNODES

*Lymphnodes of the head and neck.* These are usually described together for convenience and include the following anatomical groups of lymphnodes, viz., the pre auricular (or parotid) group, posterior auricular (or mastoid) group, occipital group, submental group, submaxillary group, superficial cervical group (under the skin and along the external jugular vein in the posterior triangle), superior deep cervical group (underneath the sternomastoid and along the internal jugular vein), and inferior deep cervical (or supraclavicular) group. The so-called tonsillar gland belongs to the superior deep cervical group.

### METHOD OF EXAMINATION

*Inspection* of the neck is first carried out from the front and sides for visible lymphnode enlargement. *Palpation* of the lymphnode is then carried out from the back of the seated patient, the two hands of the standing examiner being placed on either side of the neck. By slow, gentle, up and down or rotatory movement of the palpating fingers, the various groups of lymphnodes are palpated systematically and compared bilaterally for size, shape, consistency, number, matting, mobility and adherence to skin.

Certain diagnostic pitfalls are worth noting. Loss of weight or fat may make normal lymphnodes more readily palpable and hence likely to be taken for enlarged nodes. Other swellings in the neck (e.g., carotid body tumour, cysts and salivary gland enlargements) may be mistaken for enlarged lymphnodes. Important cervical glands, situated behind or underneath the sternomastoid, may be missed completely unless specially looked for, after relaxing the sternomastoid by pushing the head to one side. Supraclavicular glands,

not felt during recumbency, can be rendered palpable by making the patient adopt the sitting-up or standing posture. Submaxillary and submental glands are best felt bimanually, with one hand (of the examiner) in the mouth and the other below the lower jaw.

**Acute inflammatory nodes (or acute lymphadenitis)** Acute inflammatory nodes of the neck are usually secondary to some local septic infection of the skin, scalp (e.g. pediculosis capitis, wounds, suppurating cysts or tinea), tonsils, teeth, gums or ears. A thorough examination of the scalp, skin of the face and neck, mouth, and throat is therefore essential. The affected nodes rapidly become enlarged, painful, tender, fixed and matted, whilst the overlying skin becomes hot, red and oedematous or brawny. This stage may be followed by resolution, chronicity, abscess-formation or even cellulitis.

**Chronic inflammatory nodes (Chronic lymphadenitis)** Common in children, this condition is usually secondary to acute lymphadenitis. There are chains of small, painless, non-tender, fixed or matted lymphnodes, either in the anterior triangle (frequently secondary to septic teeth, gums or adenoids) or in the posterior triangle (secondary to a septic condition or pediculosis of the scalp).

**Tuberculous lymphadenopathy** Tuberculous infection of the neck, common in debilitated or undernourished children and young adults, usually starts in an upper cervical node behind the angle of the jaw but may first implicate the preauricular, posterior auricular, submaxillary or supraclavicular group of nodes, later involving some or all of the deep cervical nodes. It may or may not be associated with tuberculous infection of lymphnodes elsewhere in the body (e.g. axillary, inguinal, tracheobronchial or intra-abdominal). In the early stages, enlarged tuberculous nodes are somewhat firm, freely moveable, discrete and non-adherent. After a while (because of caseation and peradenitis), they become softer, immobile, matted together and adherent to skin and adjacent structures. In the later stages, healed or draining skin sinuses, calcification, cold abscess formation and sepsis complicate the picture.

**Syphilitic lymphadenopathy** Although possible in any of the three stages of syphilitic infection, cervical lymphnode involvement is the commonest and most characteristic in the secondary stage, with its generalized lymphadenopathy and rash. The cervical lymphnodes, most involved in *secondary syphilis* are the occipital, posterior auricular and epitrochlear, they are characteristically firm, discrete, "shotty", almond-shaped, painless, non-adherent to skin and adjacent tissues, non-suppurating and usually last for weeks or months. Appearing simultaneously and confirmatory of the diagnosis is the syphilitic rash or roseola (a pleomorphic, symmetrical, non-itching, "raw-ham" coloured or "coppery" rash), most marked on the forehead, the "corona veneris", and over the trunk, flexor surfaces and creases of the body.

In *primary syphilis*, rapid development of large, fleshy, soft or firm lymphnodes in the neck, in association with an indurated extra-genital chancre on the lip, face or tongue, is characteristic. In the *tertiary stage*, gummatous involvement of cervical nodes, which is rare, may lead to large, soft or elastic, adherent lymphnodes, later ulcerating into a typical gummatous ulcer.

**Sarcoidosis** Cervical lymph nodes may be enlarged with bilateral hilar lymph node enlargement. Cough, weight loss and erythema nodosum are common associated symptoms.

**Hodgkin's disease (Fig 6.5)** A slow-growing, massive enlargement of lymphnodes usually starting on one side of the neck (particularly involving the nodes underneath the middle third of the sternomastoid and the supraclavicular group) in children or young males in association with anaemia,

enlargement of lymphnodes elsewhere (e.g., axillary, inguinal or mediastinal nodes), enlarged spleen and liver, increasing debility, pyrexia and lymphocytosis or eosinophilia, should suggest the possibility of lymphadenoma. The lymphnodes, which may remain confined to the neck for years, are typically elastic in consistency, discrete, large, moveable, usually non-adherent to skin and deeper structures, non-suppurative and frequently arranged as a large conglomerate mass ("bunch of grapes" appearance).

**Non Hodgkin lymphomas** This covers a wide variety of lymphomas formerly classified as lymphosarcoma, reticulum cell sarcoma, giant follicular lymphoma, etc. Though clinically resembling Hodgkin's disease in some respects, these do not contain Reed-Sternberg cells, show a wide variation in malignancy, there is a far less orderly progression of disease, and unlike Hodgkin's these lymphomas have a predilection for such sites as the gastrointestinal tract and CNS.

**Lymphatic leukaemia** The enlargement of the cervical lymphnodes, in this disease, is usually of minor order and part of a generalized adenopathy, associated with a diagnostic blood picture and tendency to haemorrhagic phenomena, increasing debility and death.

**Lymphadenopathy in rubella** Rubella or German measles may be suggested, especially during times of epidemics, by a sudden enlargement of occipital and posterior cervical lymphnodes, in association with fever, cold and morbilliform rash.

**Lymphadenopathy of infectious mononucleosis** The sudden appearance of enlarged cervical nodes in association with similar nodes elsewhere, in a child or young adult with fever and sore throat, should be sufficient indication for special blood investigations to rule out glandular fever.

**Secondary or metastatic lymphnode enlargement in the neck** is secondary to malignancy (e.g., carcinoma or melanosarcoma) of the lip, tongue, gum, face or scalp.

**Drug-induced lymphadenopathy.** Various drugs may give rise to lymphnode enlargement. They include carbamezapine, iron dextran, meprobamate, PAS, phenylbutazone, troxidone, etc. Diphenylhydantoin may cause not only lymphadenopathy but also hepatosplenomegaly.

**Significance of individual lymphadenopathies** Exact localization of lymphnode enlargement in the neck may be of considerable informative value. Thus *preauricular* or *parotid* lymphadenopathy may be due to septic infections of the skin of the eyelid, cheek, temple or ear, lupus vulgaris, rodent ulcer, epithelioma or tuberculosis. *Posterior auricular* or *mastoid* lymphadenopathy suggests sepsis of the scalp or side of head, *Occipital* lymphadenopathy (near the insertion of the trapezius muscle) suggests sepsis, seborrhoea, wounds or pediculosis capitis of the back of the scalp, rubella, secondary syphilis or leukaemia.

*Submental* and *submaxillary* lymphadenopathies suggest infections of the lip, tongue, face, teeth, gums and floor of mouth, epithelioma or tuberculosis. The *superficial cervical* nodes may be enlarged in septic infections of the scalp or hair. The *tonsillar* gland (just behind the angle of the jaw) may enlarge in cases of septic tonsillitis, quinsy, Vincent's disease, diphtheria, tuberculosis, syphilis or epithelioma. The *superior deep cervical* nodes (underneath the sternomastoid) may be involved in tuberculosis, lymphadenoma, lymphosarcoma, secondary deposits or sepsis of the face, mouth, nose, head, scalp, pharynx, trachea or larynx.

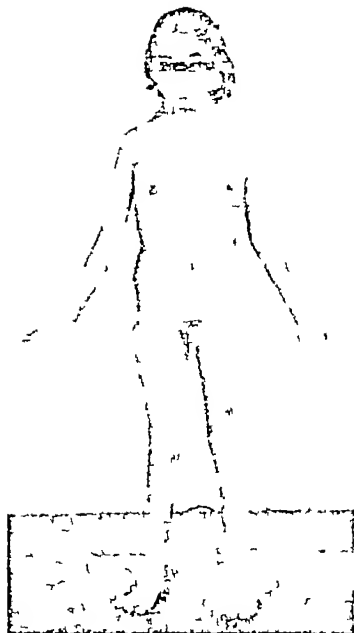


Fig 61 Webbed neck, stunted growth syndrome in a girl of 17 years having normal sexual development and menstrual periods

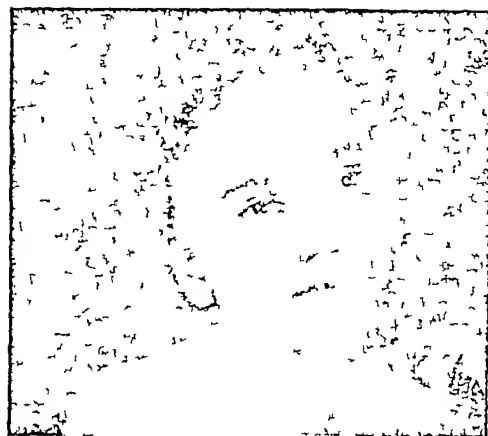


Fig 62 Congenital torticollis



Fig 64 Cellulitis of neck (bull neck) in diphtheria

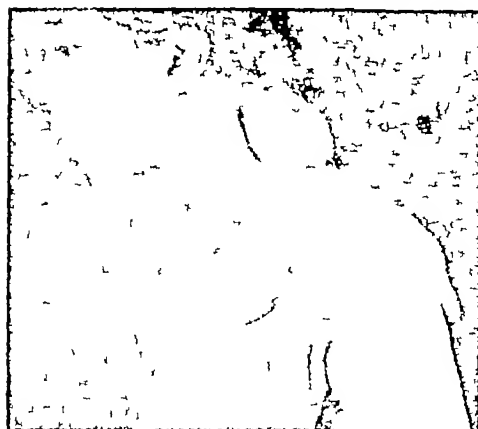


Fig. 6.3 Pedunculated neurofibromatous tumour of neck

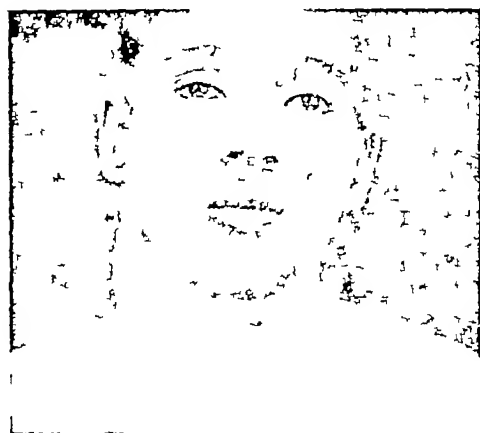


Fig 65 Cervical lymphadenoma

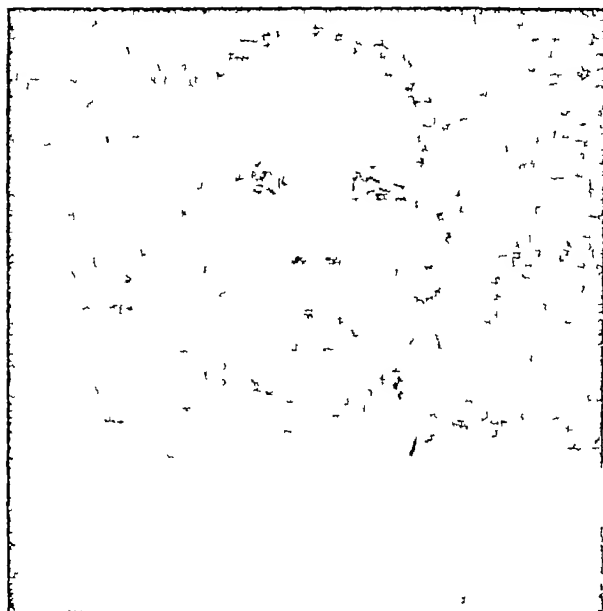


Fig 66 Virchow's node in carcinoma of stomach



Fig 67 Simple goitre



Fig 68 Endemic goitre

Fig 69 Cold tumour

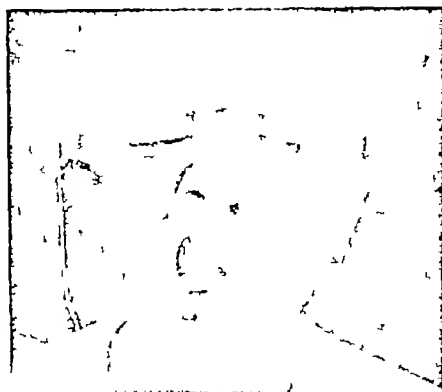
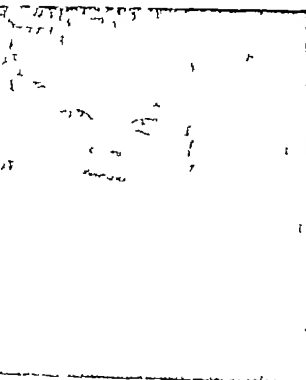


Fig. 610 Malignant tumour of parotid with facial paralysis

Fig. 611 Teratoma of neck

The *inferior deep cervical* or *supraclavicular nodes* (just above the clavicle near the insertion of the sternomastoid) may be enlarged in tuberculosis, lymphadenoma, lymphosarcoma, malignancy of stomach, lung or oesophagus, and infections anywhere on the head, neck, arm, breast or upper part of chest

**Virchow's nodes** (Sentinel node, signal node) (Fig 6 6) A small fixed lymphnode in the left supraclavicular fossa, near the inner end of the clavicle, and highly suggestive of malignancy of the stomach, kidney or testicle A similar gland on the right side may be noted in cases of intrathoracic new growth (usually of the lung or oesophagus)

## THE THYROID GLAND

**Anatomy** The thyroid gland is composed of two lateral lobes on either side of the trachea, and an isthmus connecting the two lobes at the level on the second, third and fourth rings of the trachea A pyramidal lobe (a linear projection running up from the left of the isthmus), retrolaryngeal lobules (behind the larynx) and accessory thyroids (either attached to a lobe or along the line of the thyroglossal duct) may also be present in rare cases

### METHOD OF EXAMINATION

**Inspection** of the neck is carried out first with the head extended and the patient carrying out movements of deglutition (by swallowing mouthfuls of water) Any swelling in the region of the thyroid gland which moves up and down with deglutition must be regarded as a thyroid swelling Abnormalities of size, shape, symmetry, surface and mobility (with deglutition) of the thyroid, as well as nodules can be perceived by inspection

**Palpation** is then carried out from behind the seated patient, whose head is held midway between flexion and extension The examiner (standing) palpates the thyroid, with the fingers of the two hands over the two sides of the gland, the thumbs resting over the back of the neck Each lobe of the gland is palpated for size, shape, asymmetry, consistency, surface, nodules, pulsations and thrills The posterior border of each lateral lobe must be palpated after displacing it laterally (by digitally pushing the contralateral lobe towards the midline) and relaxing the sternomastoid muscle In this way hidden nodules or swellings on the posterior aspect can be brought to light The isthmus of the gland is then palpated in the midline and the pyramidal lobe looked for along its upper border The lower border of the thyroid is then palpated (during deglutitional movements) with a view to excluding a retrosternal prolongation of goitre, a condition capable of causing either pressure symptoms or signs of thyrotoxicosis on its own The trachea and larynx are then palpated for pathological displacement or compression

**Auscultation** over the gland may disclose a continuous or systolic murmur, indicative of thyrotoxicosis

Accessory methods of investigation, such as transillumination of the gland to distinguish solid from cystic lumps, percussion over the manubrium sterni for evidence of retrosternal

goitre, radiological examination for retrosternal shadows and tracheal displacement or compression ("scabbard trachea"), laryngoscopy, and physical examination for signs of pressure or thyrotoxicosis, may be needed in order to make the examination of the thyroid gland complete

## INTERPRETATION

In the examination of the thyroid gland, certain features of diagnostic importance are worth noting. A normal gland may appear enlarged in a thin or wasted individual. Physiological thyroid enlargements are common during menses, pregnancy and puberty. A thyroid swelling which fails to display up and down movement on swallowing suggests either malignancy, chronic thyroiditis, an impacted retrosternal goitre or a massive or heavy enlargement. A non-thyroid swelling (such as subhyoid bursa, or perichondritis of the laryngeal cartilage) may show an up and down movement with deglutition on rare occasions. Occasionally, a large fatty pad in the supra-sternal notch may simulate a thyroid swelling. Thyrotoxicosis may be associated with a perfectly normal-sized gland, especially in the event of a retro-sternal extension. A massively enlarged thyroid is seldom toxic (the majority of toxic goitres being small or medium-sized). A normal thyroid may appear prominent or enlarged in cases of neoplasm or aneurysm situated behind the sternum at the root of the neck.

**Goitre** The word "goitre", although restricted at times to non-inflammatory enlargements of the thyroid gland only, may rightly be applied to any form of thyroid gland enlargement, either inflammatory or non-inflammatory. A goitre may or may not be associated with signs of thyrotoxicosis, in fact, massive goitres are usually non-toxic.

**Types of goitres (or thyroid enlargements)** The various enlargements of the thyroid gland are considered here.

**Simple goitre** (Fig 6.7) (Simple parenchymatous goitre, simple non-toxic goitre. Colloid goitre, endemic goitre) (Fig 6.8) Sporadic or endemic, common in hilly districts (of Switzerland, Derbyshire, Great Lakes and Northern India), and caused by iodine deficiency, the simple goitre is usually smooth, soft, diffuse, massive, non toxic, non adherent and associated (quite frequently) with pressure symptoms.

**Nodular goitre** (Simple adenomatous goitre, non toxic adenomatous goitre) The thyroid swelling is characteristically nodular or irregular, elastic in consistency, non adherent to skin and tissues, and non toxic. It has a tendency, however, to become toxic or even malignant after years.

**Exophthalmic goitre** (Graves' disease, Basedow's disease, primary thyrotoxicosis) The goitre is usually small or medium-sized, smooth, diffuse, symmetrical, non adherent and associated with toxic signs from the very start.

**Toxic adenoma** (Toxic adenomatous goitre, secondary thyrotoxicosis, secondary hyperthyroidism) The condition is identical to that of "nodular goitre", except for the presence of characteristic signs of thyrotoxicosis. A nodular goitre, non toxic for years, may become toxic ("toxic adenoma"), due to oversecretion of thyroxine by one or more of the nodules.

**Malignant goitre (Adenocarcinoma of thyroid)** A rapidly growing, irregular, somewhat spherical, nodular, fixed or immobile, usually non-toxic, stony-hard, adherent enlargement of the thyroid gland, associated with pressure symptoms (like stridor, cough, dyspnoea and hoarseness of voice) and early metastatic deposits (in regional lymphnodes, brain and bones) It is usually superimposed on a long-standing nodular goitre or toxic adenoma

**Riedel's disease ("Woody" thyroiditis)** Common in middle-aged or elderly women and of unknown origin, the thyroid swelling of Riedel's disease is usually very hard or "woody" painful, diffuse or unilateral, tender, smooth, adherent to deeper structures but not to skin, non-toxic and associated with early pressure symptoms

**Hashimoto's disease (Lymphadenoid goitre)** Common in obese, elderly women and of obscure origin, the thyroid swelling is always diffuse or bilateral, firm but not hard, smooth, mobile, non-adherent to skin and deeper tissues, non-toxic (or even associated with hypothyroidism) and with few or no pressure symptoms

**Retrosternal goitre** This may be either in the form of a retrosternal projection or prolongation of an ordinary goitre, a "plunging" goitre (visible or palpable only on swallowing or violent coughing) or an entirely intrathoracic or retrosternal goitre Although liable to be missed, it may cause serious pressure symptoms, thyrotoxicosis without a visible goitre, engorged neck veins, manubrial dullness (simulating a tumour or aneurysm) or radiologically, a perplexing "rounded shadow" in the chest simulating an intrathoracic tumour (but mobile on deglutition)

**Benign metastasizing adenoma (Malignant adenoma)** An uncommon form of low-grade malignancy of the thyroid gland associated with metastatic deposits, but where both the adenoma and the deposits contain normal thyroid tissue

**Physiological goitre** A simple and transitory enlargement of the thyroid gland occurring at the time of pregnancy, puberty, menses or menopause It is of no significance

**Hypothyroid goitre** An enlargement of the thyroid associated with a state of hypothyroidism (e.g., myxoedema or cretinism)

**Aberrant goitre** A rare form of goitre encountered at the root of the tongue (lingual goitre) or situated anomalously near the hyoid, or within the trachea, oesophagus or supraclavicular fossa

## THE SALIVARY GLANDS

**Enlargements of the salivary glands** The main causes of enlargement (particularly of the parotid and submaxillary glands) are a *salivary calculus* (causing intermittent swelling or a sialoadenitis), a *simple non-suppurative inflammation*, usually a parotitis, with sudden, unilateral tender enlargement of the gland, associated with high fever, malaise, redness and oedema of the overlying skin, and usually secondary to bad oral hygiene, especially after abdominal operations and in states of debility, so-called *mixed tumour* usually of the parotid (Fig 6 9) or submaxillary gland and characterized by a slow-growing, nodular or rounded, painless and moveable swelling, which may become malignant after many years, *malignant tumour* (Fig 6 10) affecting any one of the salivary glands, to the nature of a carcinoma or sarcoma and causing a rapid and painful enlargement with extreme hardness and immobility of the gland, infiltration of the skin and deep tissues, paralysis of the facial nerve and regional

lymphnode involvement, *epidemic parotitis* or *mumps*, an acute infectious disease, mainly of children, affecting both the parotids, one after the other, and at times, submaxillary and sublingual glands, testis, ovary and pancreas, the parotids usually remain painful, tender, swollen and firm for one to two weeks, *von Mikulicz's syndrome*, a rare form of slow, symmetrical, painless swelling of parotid, submaxillary and lacrymal glands on both sides, occasionally associated with a blood picture of lymphatic leukaemia with enlargement of lymphnodes and spleen

Bilateral enlargement of parotid glands may be seen occasionally in cirrhosis of liver or other types of malnutrition such as kwashiorkor

## CYSTS

Cysts are common in the neck region. They may be congenital or acquired. The following varieties require consideration

**Thyroglossal cyst** A small, subcutaneous, spherical, midline cyst close to the hyoid bone and with a tendency to form a thyroglossal fistula after suppuration. It is due to persistence of a part of the thyroglossal duct.

**Branchial cyst** A slow-growing, painless, spherical or ovoid cyst between the anterior border of the sternomastoid and the hyoid or thyroid cartilage. It is due to persistence of part of the third branchial cleft.

**Dermoid cyst** A small, painless, cystic swelling in the midline of the neck, not attached to skin or deeper structures. It is often called a "sequestration dermoid".

**Cystic hygroma (Lymphangioma)** A large, soft, painless multilocular, semi-translucent cyst containing lymph, at the root of the neck in children. A unilocular cyst of this nature is called a serous cyst.

**Pneumatocele** A herniation of the lung apex into the root of neck, after injury or emphysema, giving rise to a soft, rounded hyperresonant swelling, with crepitus on squeezing and an expansile impulse on coughing or straining. A small cystic swelling with similar characteristics (aerocele) containing air may protrude from the trachea or thyroid cartilage in players of wind instruments.

**Subhyoid bursa** This appears as a small ovoid cyst, just below the hyoid, and likely to be mistaken for a thyroglossal cyst.

**Blood cyst** A cystic or angiomatous swelling, containing blood, with a venous thrill and an impulse on coughing.

**Fistulae of neck** A *branchial fistula* (or lateral fistula) of the neck presents a small opening on the skin along the lower third of the anterior border of the sternomastoid on one side. It is due to persistence of the third branchial

cleft A *thyroglossal fistula* (or median fistula) presents a small midline opening on the skin, close to the hyoid bone, it arises in conjunction with the embryonal thyroglossal duct

**Solid tumours of the neck** These may be benign or malignant A *benign tumour* of the neck may be a lipoma (single or multiple, sessile or pedunculated fatty tumour), myxoma, fibroma, osteoma or chondroma A *malignant tumour* may be of the nature of a secondary metastatic gland (secondary to carcinoma of the tongue, lip, mouth, pharynx, oesophagus, nose, ear, stomach, lung or larynx), *lymphosarcoma* (arising in a lymphnode), *carotid body tumour* (a rare chromaffin-cell-containing, and sometimes familial tumour arising in the carotid body and with a tendency to malignancy), *potato tumour* (a highly malignant type of tumour), or *teratoma* (Fig 6 11)

**Blood vessels of the neck** Engorgement and pulsations of the jugular veins and carotid arteries in the neck are of great diagnostic value They are adequately discussed in Chapter 9

#### THE TRACHEA

Examination of the trachea is usually carried out during the examination of the respiratory system The student should refer to that chapter for details of examination

# 7 | The Extremities

## THE UPPER EXTREMITIES THE SHOULDERS AND ARMS

### DEFORMITIES

**Absence of clavicles** (Fig 7 1) This may arise occasionally, congenitally, allowing the patient to approximate the two shoulders in front of the chest. Absence of clavicles is normal in horses.

**Dislocation of shoulder** Either *congenital* (unilateral or bilateral) or *acquired* (from injury or acute polyomyelitis), it shows an abnormality of position with flaccid "hanging" of the arm, limitation of movement and wasting of muscles near the shoulder.

**Sprengel's shoulder** A "winging" of the scapula, with prominence of its vertebral border and lower angle, and with limitation of movements (forward pushing or raising of the arm), may result from paralysis of the serratus magnus (with or without involvement of the trapezius and rhomboids) secondary to injuries or operations involving the long thoracic nerve.

**Congenital absence of extremity** (Fig 7 2) Whole or part of an extremity may be congenitally absent, failure of development may affect the whole arm or leg (amelus), its proximal portion (phocomelus) or its distal portion (hemimelus).

**Cubitus valgus and varus** Normally, the forearm and arm meet at an angle of about 170 degrees ("carrying angle") opening outwards. A decrease of this angle (with the hand further away from the body) constitutes cubitus valgus, and increase of it, cubitus varus.

**Madelung's deformity** A defective growth of the lower end of the radius, from trauma or overstrain, resulting in a deflection of the hand and wrist towards the outer side and front by the normal-sized ulna.

**Flail arm** An abnormal resting position of the arm resulting from acute polyomyelitis, brachial plexus injuries (upper arm type or Erb-Duchenne type), birth injuries (obstetrical paralysis) or heavy falls on the shoulder. In nerve injuries, the arm may assume the "policeman's tip position."

**Spastic arm (*hemiplegic arm*)** After hemiplegia, the arm may remain adducted and flexed at the elbow, wrist and hand joints

**Holt-Oram syndrome** The thumb is hypoplastic and in the same plane as the rest of the fingers or the fingers may be triphalangeal, absent or longer than normal

#### LOCALIZED SWELLINGS

The arms and forearms may be the seat of a large number of local swellings, lumps or nodules, e.g., rheumatic nodules, neurofibromatous lumps, polyarteritis nodosa nodules, multiple lipomata, gouty tophi, xanthomata, tumours of skin, fat, muscles, nerves and bones (benign or malignant), syphilitic, tuberculous or osteomyelitic involvement of bones, affections of joints, "ganglion" and tenosynovitis. Some of these require special consideration. The others are dealt with in surgical textbooks.

**Rheumatic nodules** (Fig 7.3) Small, discrete, painless, non-tender, subcutaneous nodules of variable size, usually bilateral, frequently associated with the tendon sheaths and common over the elbows, wrists and fingers in cases of acute rheumatic fever. Their presence usually suggests "activity" of the rheumatic state.

**Rheumatoid nodules.** The typical skin lesions of rheumatoid arthritis are the nodules which are present in perhaps a quarter of all patients. These are found in the subcutaneous tissues overlying bony points, especially around the elbow joint. They vary in size up to a diameter of 2-3 cm, their presence suggests that the disease will run an unfavourable course.

**Multiple neurofibromatosis** Small circumscribed neurofibromatous tumours under or within the skin and associated with pigmentation.

**Polyarteritis nodosa nodules.** Minute subcutaneous nodules, difficult to palpate, may be discovered occasionally along the blood vessels of the forearm or arm in cases of periarteritis nodosa.

**Gouty tophi.** Fairly common in gouty subjects, these tophi are particularly common over the tip of the elbow and knuckles of the hand (Fig 7.4) and may attain gigantic proportions. They are usually hard, painless, non-tender, subcutaneous nodules of variable size, containing sodium biurate crystals.

#### OEDEMA

Oedema of the upper extremity (Fig 7.5) or extremities may be a part of anasarca, or occur after radical breast-removal, venous thrombosis, infections of the upper extremities, malignant axillary lymphnodes, intrathoracic tumour or aneurysm, and neurological diseases such as hemiplegia.



## LYMPH NODES

**Axillary lymphadenopathy.** Enlargement of the axillary lymphnodes may be unilateral or bilateral and either confined to the axilla or part of a generalized lymphadenopathy. The important causes of enlargement are septic infections of the upper extremity shoulder, back or breast, carcinoma of breast, lymphadenoma, tuberculosis or lymphatic leukaemia.

**Epitrochlear lymphadenopathy.** Enlargement of lymph nodes above inner condyle of humerus may be indicative of sepsis of the fingers (e.g. whitlow), hand or forearm, a primary extra-genital chancre (digital) or lymphadenitis of secondary syphilis.

## JOINTS

See examination of the arthritic patient

## BONES

The long bones of the upper (or lower) extremities may reveal certain abnormalities of diagnostic importance.

**Bone tenderness.** Tenderness or pain (on tapping or pressing a bone) may be *localized* as in fractures, infections of bones and bone tumours or *diffuse* as in leukaemia, aplastic anaemia, severe macrocytic or microcytic anaemia, wasting disease or hysteria.

**Angular deformities.** These may be caused by hyperparathyroidism (osteitis fibrosa cystica), osteitis deformans (Paget's disease), osteomalacia (adult rickets), osteogenesis imperfecta (fragilitas ossium), metastatic bone deposits or badly united fractures.

**Smooth curvatures of the shafts of bones** may be due to syphilis, rickets, osteomalacia or osteitis deformans. In rickets enlargement of the epiphyses at the wrists and ankles is easily recognizable because of the thinness of the subcutaneous tissues in these regions.

**Localized swellings over the bones** may be due to acute periostitis, syphilitic nodes (periosteal nodes or gummata), cystic bone tumours of hyperparathyroidism, callus formation in fractures and benign or malignant tumours of bone.

**Spontaneous fractures.** Multiple pathological or spontaneous fractures affecting the long bones of the arms (or legs) should suggest certain possibilities like osteogenesis imperfecta, osteitis fibrosa cystica, myelomatosis, secondary malignant deposits, osteoporosis (marble bones), thymoma, Gaucher's disease and Hand-Schüller-Christian syndrome.

## ARTERIES

**Arteriosclerosis.** The state of the arterial walls can be easily ascertained from palpation of the brachial (with arms semi-flexed) and radial arteries.



Fig 71 Congenital absence of clavicles



Fig 73 Rheumatic nodules over the elbow



Fig 72 Congenital underdevelopment of upper extremity



Fig 74 Gouty tophi

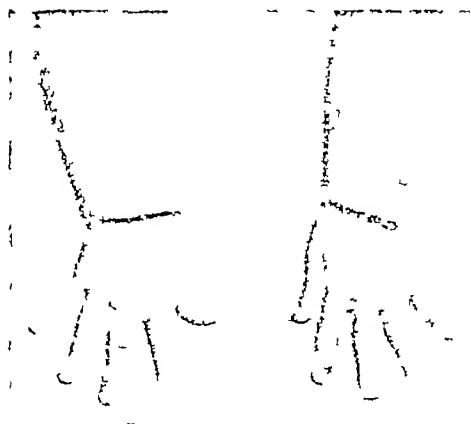


Fig 75 Unilateral oedema of upper extremity

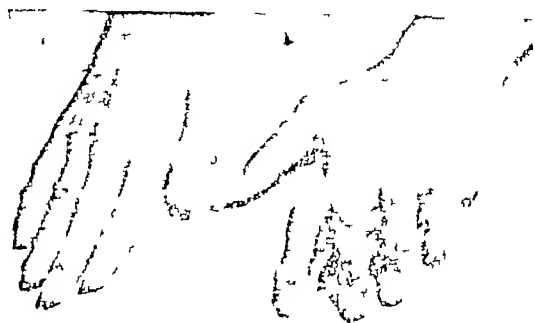


Fig 77 Unilateral claw hand (main en griffe)

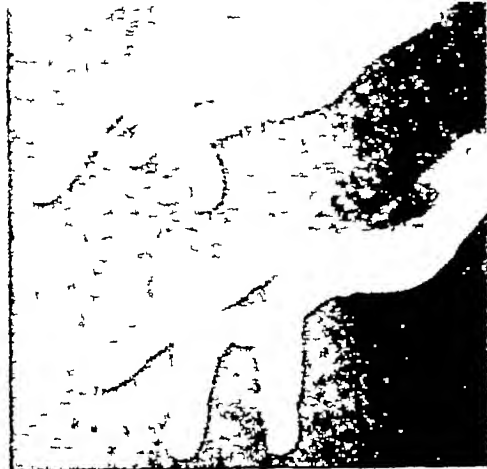


Fig 7.9 Dupuytren's co-tracture.



Fig. 7.10 Acromegalic hands compared with normal

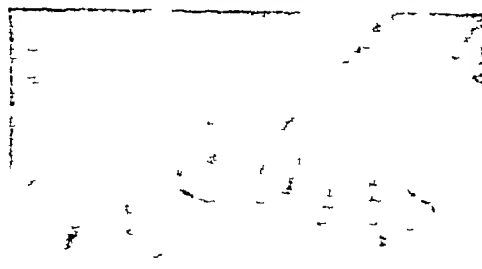


Fig. 7.11 Mass in succulente of syringomyelia

Fig. 7.12 Other hand

In case of arteriosclerosis or Monckeberg's degeneration, the arteries feel thickened, tortuous, nodular or calcified (so-called "pipe-stem" or "whip-cord" arteries)

The study of the arterial pulse is described in Chapter 9

## VEINS

The veins of the upper extremities may be engorged, collapsed or affected by inflammation

**Engorged (or distended) veins.** This may be due to congestive cardiac failure, superior vena caval obstruction from aneurysm or tumour, pressure on the arm veins from enlarged lymphnodes or malignant tumours, or to venous thrombophlebitis

**Collapsed veins** This may occur in cases of shock, diabetic coma, or dehydration. It may offer a serious impediment to the much needed intravenous therapy in such cases

**Venous thrombosis** A condition affecting the deep or superficial veins of the upper extremities, secondary to infection, intravenous injection, trauma or excessive strain. The characteristic findings are a tender and painful cord-like thickening of the vein affected, associated with oedema and cyanosis of the distal part and engorged collateral veins over the arm

## THE HAND

The presence of a hand is the prerogative of the human race. All other mammals have either a front paw or foot.

**State of temperature** *Warm hands* are common in fevers, thyrotoxicosis, rheumatoid arthritis, erythematous states and at times in normal individuals. *Cold hands* are common in neurasthenia, neurocirculatory asthenia, anxiety, shock or peripheral failure, vasovagal attack, collapse, profuse haemorrhage, during rigors, Raynaud's syndrome and peripheral vascular diseases

**State of moisture** *Excessive dryness* of the hands may be noted in fevers, dermatitis, wasting diseases, myxoedema, cretinism, dehydration, ichthyosis or xeroderma. *Excessive moisture* of the hands ("sweaty palms") may occur in thyrotoxicosis, nervousness, neurocirculatory asthenia, vasovagal syndrome, shock, vasomotor neurosis or rheumatoid arthritis

**Colour.** The colour of the hand (particularly of the palm) may yield useful information. Thus, it may be pale in anaemia, oedema or myxoedema, purplish in polycythaemia, bluish in cyanosis, yellow or orange in carotinaemia, jaundice and xanthomatosis, and bright red in palmar erythema or liver palms

**Palmar erythema (liver palms)** (Fig. 7 6) Here the hands are warm, the palms bright red in colour, especially the hypothenar and thenar eminences

Islets of erythema may be seen at the bases of the fingers. The soles of the feet may be similarly affected. Causes—Familial, cirrhosis of liver, rheumatoid arthritis, pregnancy, chronic febrile diseases, chronic leukaemia, thyrotoxicosis. Palmar erythema in cirrhosis is due to an extensive collection of arteriovenous anastomosis. A painful type of palmar erythema has been described in dry beri beri. A form of palmar erythema has been found in patients who demonstrated the shoulder hand syndrome following myocardial infarction. About 3 to 5 per cent of normal individuals have palmar erythema. Alterations of colour may also be due to exanthematous fevers (like measles, rubella and scarlet fever), vitamin deficiencies (e.g., pellagra), vascular diseases (e.g. Raynaud's syndrome), endocrine diseases (e.g., Addison's disease) or local skin diseases (like eczema or dermatitis).

**Handshake** Besides affording a clue to the mental or emotional make-up of the individual, the manner of shaking hands may suggest certain diseases. The violent handshake of the maniacal patient, the subdued handshake of the melancholic, the interrupted or suddenly withdrawn handshake of the schizophrenic, the clumsy handshake of the mongol, the prolonged or persistent handshake (with slow relaxation of the grasp) in myotonia atrophica, *the tremulous and stiff handshake of the Parkinsonian, are all characteristic in their own way*.

**Handwriting** Inability to write may be due to a central nervous lesion (motor agraphia), peripheral nerve lesions, loss of power or spasticity of muscles, wasting of muscles, painful joint affections, bony injuries, mental defect or writer's cramp.

Characteristically abnormal handwriting may be noted in a variety of diseases, e.g., schizophrenia, general paralysis of the insane, hyperthyroidism and chorea. In schizophrenia, the writing may be full of symbols, diagrams, new words, repetitions and fantastic conceptions. In G.P.I., the letters and words may be uneven in size and shape with uneven spacing and blots and scratches. In thyrotoxicosis, the writing may be tremulous and full of mistakes, some letters being obliquely written or even inverted.

**Outstretched hand** A mere inspection of the outstretched hand or hands may disclose the typical choreiform posture, with hyperextension of the metacarpophalangeal and interphalangeal joints and flexion of the wrist-joint in chorea, so-called "piano-playing" movements of the fingers in tabes dorsalis (with the eyes closed) and due to defective maintenance of posture, fine, medium or coarse tremors in a variety of disorders, or a tendency to unilateral deviation of the hand in cerebellar disease.

**Abnormal movements** The hand or fingers may be the seat of a variety of abnormalities of motion such as tremors, writer's cramps, choreiform movements, athetotic movements, carpal spasms of tetany, carphology (picking of bed clothes, as in high fevers or prostration of status typhosus), subsultus tendinum (play of tendons of the hands or wrists), Jacksonian fit and epileptiform convulsions.

**Hyperkeratosis palmaris et plantaris (tylosis)** In this condition gross thickening of palms and soles occurs as an autosomal dominant trait. Tylosis palmaris is also known to occur in carcinoma of oesophagus.

**Inability to relax the grip** It is nearly always due to myotonia. It rarely occurs in association with a lesion of the contralateral frontal lobe of the brain, and is due here to tonic preservation of the grip due to slow relaxation.

**Unequal size of hands** Inequality in the size of the two hands may be due to unilateral oedema of one hand (from thrombophlebitis, radical mastectomy, local obstruction to blood flow or gravitationally in any variety of oedema), acute anterior poliomyelitis, congenital hemihypertrophy, or unilateral wasting of hand muscles.



Fig 77A Palmer erythema in cirrhosis of liver



Fig 719 Thromboangitis obliterans with gangrenous ulcers

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## DEFORMITIES AND CONTRACTURES

**Main en griffe** (Griffin hand, claw hand) (Fig 7 7) A characteristic claw-like attitude of the hand with flexion of the interphalangeal and extension of the metacarpophalangeal joints may be secondary to weakness or paralysis of the interossei and lumbrical muscles permitting overaction of the extensor communis digitorum and flexor digitorum muscles. The important causes of a claw-hand are ulnar nerve paralysis (the ring and little fingers being mainly affected), progressive muscular atrophy, syringomyelia, amyotrophic lateral sclerosis, median nerve paralysis, brachial plexus injury, leprosy, and, rarely, cervical rib, cervical pachymeningitis or acute poliomyelitis.

**Ape hand** A deformity of the hand characterized by the thumb falling in line (or on a plane) with the fingers. It may arise in progressive muscular atrophy, amyotrophic lateral sclerosis or syringomyelia (from involvement of the abductor pollicis muscle).

**Preacher's hand** Hyperextension of the wrist (as the result of early involvement of the flexor muscles of the wrist), in some cases of progressive muscular atrophy or syringomyelia, gives rise to this deformity.

**Accoucher hand** (Obstetrical hand) (Fig 7 8) A characteristic "conelike" attitude of the hand, with the fingers extended at the interphalangeal and semi-flexed at the metacarpophalangeal joints and closely approximated together, the thumb is strongly abducted and opposed to the palm or middle finger. The accoucher hand may arise spontaneously (during "carpopedal spasms") in tetany, or may be artificially induced in latent tetany (Trousseau's sign). It may rarely occur in athetosis or hysteria.

**Wrist-drop** A characteristic attitude of excessive flexion at the wrist-joint with inability to raise the hand may be due to lead poisoning, radial nerve paralysis, poliomyelitis, arsenical or alcoholic neuritis.

**Dupuytren's contracture** ("Position of Papal benediction") (Fig 7 9) This is a thickening of the palmar fascia, which contracts and causes flexion of the fingers, the ring and little fingers are the first affected. The joints of the individual fingers may become ankylosed. Dupuytren's contracture produces a hard knotty thickening and the overlying skin is puckered. It may occur in otherwise normal persons, in about a quarter of alcoholic cirrhotics, as a sequel of the shoulder-hand syndrome, and in association with idiopathic epilepsy.

**Holt-Oram syndrome** Here there is maldevelopment of thumb or first metacarpal bone. The thumb may resemble a finger and have three phalanges rather than two. The fifth finger may be missing and there may be radioulnar synostosis.

**Volkman's ischaemic contracture** A characteristic deformity of flexion of the wrist and interphalangeal joints, extension of the metacarpophalangeal joints and pronation of the hand. It is usually due to tight or faulty splinting or bandaging of the arm resulting in defective blood supply to the forearm muscles.



**Club hand** A developmental anomaly of a "stump-like" or malformed hand, secondary to non-development of muscles, or absence or deficient growth of the radius or ulna (probably secondary to a defective limb bud)

#### CHARACTERISTIC TYPES OF HANDS

**Cretinoid hand** In cretinism, the hand may show a characteristic square palm, short, fat and blunt fingers and a short radius bone

**Mongoloid hand** In mongolism or mongolian idiocy, the hands are short and thick, with short thumbs arising from the palms at levels lower than normal and with the little fingers curiously bent or curved in their mid-portions

**Paw-hand** ("Acromegalic hand", spade hand) (Fig 7 10) In acromegaly, the hand is usually massive, and "paw-like" with pad-like thenar and hypo-thenar eminences, fat, cylindrical and spatulate fingers with blunt tips and broad or square nails

**Eunuchoid hand** In hypogonadism, the hand may be long, narrow and thin skinned, with delicate and tapering fingers. A long and tapering hand may also be noted in other diseases like arachnodactyly or hyperthyroidism and in normal individuals

**Acromicria** Minute or tiny hands may be due to infantilism or developmental anomaly, usually the cause remains obscure

**Spade-hand** Non-bony thickening of the tissues may result in a thick and spade-like hand in cases of myxoedema and in acromegaly (where bony changes are associated)

**Trident hand** In achondroplasia, the fingers (and toes) are abnormally divergent like the spokes of a wheel, the middle finger usually being no longer than the others. The hands are usually short, broad and fatty

**Mam en succulente** (Fig 7 11) The "succulent hand", with sausage-like fleshy swelling of the fingers, thickening of the skin and subcutaneous tissues, trophic lesions and wasted hand muscles, is highly suggestive of syringomyelia

**Milkmaid's grip** (Grip sign) In rheumatic chorea making the patient squeeze the examiner's hand results in variation of pressure characterised by sudden releases alternated with renewed contractions

**Broad hand** Stubby fingers, clinodactyly (curving of the little finger radially), claw hand and flexion contractures may be seen in Hurler's syndrome

**Podgy hand** Swollen oedematous appearance of hands with coarse dry skin in myxoedema.

**Oath hand** (Fig 7 12) A lesion of the median nerve at the elbow or more proximally causes paralysis of long finger flexors with the exception of flexor carpi ulnaris. If the patient is asked to flex his fingers, the so-called 'oath hand' position results

**Square hand** In osteoarthritis involving the hand, bony swelling of the carpometacarpal joint of the thumb, adduction of the thumb metacarpal, and associated wasting of the small muscles produces the 'square hand' appearance

**Dimpling (Knuckle sign)** Shortening of the metacarpals usually fourth and fifth, in pseudohypoparathyroidism produces a characteristic dimpling when the patient makes a fist

## FINGERS

**Polydactyly** A rare condition of extra or supernumerary fingers or toes either congenital, familial or associated with certain diseases (such as ventricular septal defect, Lawrence-Moon-Biedl syndrome and Turner's syndrome) A record number of 37 fingers and toes has been reported in such a case

**Syndactyly (Webbed fingers)** This is an affection of one or several fingers, the fusion between the adjacent fingers being dermal or osseous It is usually seen with multiple gross congenital abnormalities though it also occurs in otherwise normal persons

**Arachnodactyly (Spider fingers)** (Fig 7 13) In Marfan's syndrome, the fingers and toes may be unduly long and thin, like a spider's legs In Marfan's syndrome when the patient grasps his own wrist proximal to the styloid process of the radius and tries to encircle it, the thumb protrudes past the ulnar side of the hand He can also encircle his wrist by grasping it with the fifth finger and thumb of the other hand (*wrist sign*) Long and tapering fingers may also occur in cases of hypogonadism, hypopituitarism, hyperthyroidism and in normal individuals

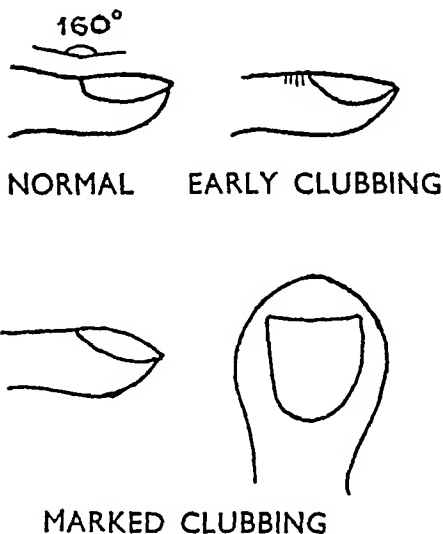


Fig 7 14

Absence of one or more fingers, or short stumps in place of fingers may be observed congenitally Macroductyly, microductyly, and brachyductyly refer to undue largeness, smallness and shortness of fingers The thumb may be absent in Fanconi's congenital aplastic anaemia

**Sausage fingers** Thick and fleshy fingers may occur in cases of syringomyelia, acromegaly or myxoedema

**Clubbing of fingers (Hippocratic fingers, serpent's head deformity of fingers)** It usually comes to the physician's notice as a physical sign, but the patient may actually complain that the ends of his fingers are swollen

*Grades of clubbing* Grade 1—There is increased glossiness and cyanotic tinge of the skin at the root of the nails Grade 2—There is increased ballotability or fluctuation of the nail in its bed consequent upon spongy thickening of the subungual tissue Abnormal mobility of the nail may be elicited by placing the nail of one examining forefinger on the base of the nail immediately above the skin fold of the finger examined and the nail of the other forefinger at the tip of the patient's nail A rocking movement may thus be imparted to the nail In presence of marked fluctuation, palpation of the nail itself may give the impression of it floating on its bed The angle of the nail bed with respect to the phalanx (normal about  $160^\circ$ ) is increased (Fig 7 14) This is clearly demonstrated by viewing the side of the flexed distal index finger or thumb (profile sign) Grade 3—There is bulbous swelling of the finger ends and excessive curvature of the nails in both planes giving a parrot beak (or watch glass) or drum stick or serpent head appearance (Hippocratic fingers) (Fig 7 15) Grade 4—Bony changes involve the wrists and ankles and at times elbows and knees (pulmonary osteoarthropathy) (Fig 7 16) The mechanism of clubbing is unknown but it is associated with increased blood flow As a rule the degree of soft tissue swelling tends to follow the severity of the primary disease Clubbing may disappear completely with recovery

#### *Causes of clubbing*

(1) *Symmetrical* (a) Acquired (i) Pulmonary Pleural, mediastinal or pulmonary disease due to compression, infection, foreign body or neoplasm Rarely in tuberculosis, congenital cystic disease, chronic passive congestion and pulmonary endarteritis (ii) Cardiac Cyanotic congenital heart disease, subacute bacterial endocarditis and occasionally congestive failure Rarely in acyanotic congenital heart disease and myxoid tumour (iii) Liver Cholangiolitic or haemochromatotic cirrhosis Rarely in portal cirrhosis and amyloidosis (iv) Gastrointestinal Conditions accompanied by chronic diarrhoea Rarely neoplasms and ascariasis (i) Miscellaneous (a) Myxoedema particularly iatrogenic, exophthalmic ophthalmoplegia (b) Hereditary (c) Idiopathic Cases without family history or recognizable underlying disease (2) *Unilateral* Aneurysmal dilatation of aorta and its branches, brachial arteriovenous fistula, Pancoast tumours, erythromelalgia and lymphangitis (3) *Unidigital* May be hereditary if bilateral and involving the thumbs Other cases result from median nerve injury, local trauma, tophaceous gout, sarcoidosis (4) *Differential clubbing* The toes may be clubbed independently of the fingers in PDA with reversed blood flow

*Macroductyly* Hypertrophy of one or several fingers may be a manifestation of Paget's disease, neurofibromatosis, or a local arterio-venous fistula (in which case the whole hand may be large)

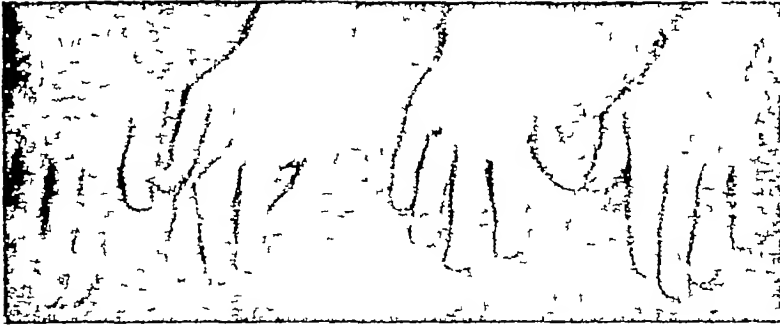


Fig 7 13 Spider fingers of arachnodactyly, compared with normal fingers

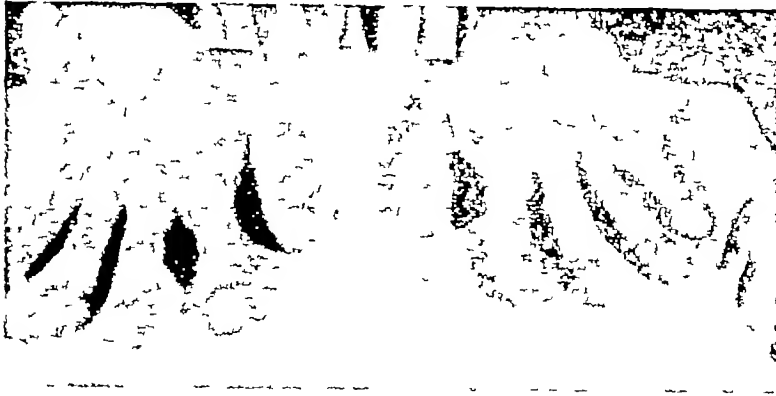


Fig 7 15 Clubbed fingers



Fig 7 16 Pulmonary osteoarthropathy

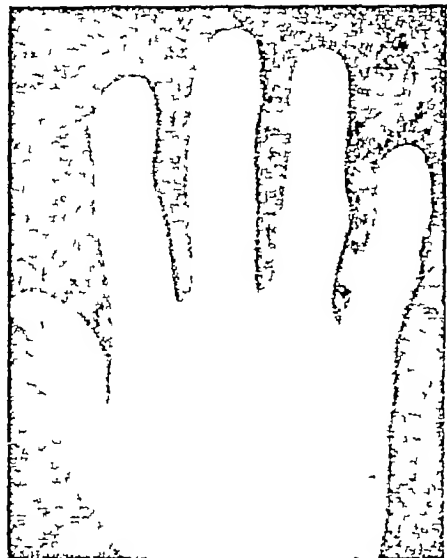


Fig 7 17 Heberden's nodes

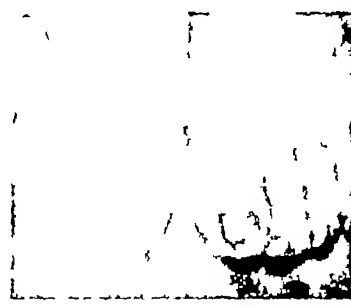


Fig 7 20 Tinea unguium

Fig 7 19 shows phenomenon in  
gangrene

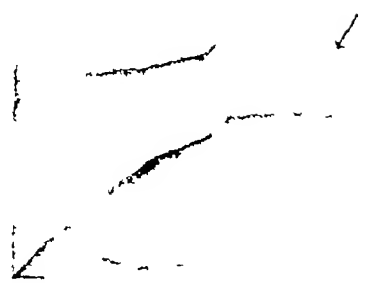
Fig 7 19 see after Fig 33

Fig 7 21 Beau's line



Fig 7 22 Leukonychia (spoon nails)

Fig 7 22A Splinter haemorrhage in subacute bacterial  
endocarditis (note Osler's node  
marked by arrow)



**Brachydactyly.** Shortened fingers which result from an absence of one of the phalanges, are an inherent dominant trait. Short fingers of equal length are a feature of achondroplasia. The fingers also look short in pseudohypoparathyroidism, where there is shortening of the metacarpals and carpals. A shortened fourth metacarpal, best noticed as a short knuckle when making a fist, is a feature of Turner's gonadal dysgenesis. The fingers of cretins appear short and broad. In hyperparathyroidism the fingers appear stubbed with bulbous ends due to collapse of distal phalanges.

**Clinodactyly.** An incurving of the fifth finger can be inherited as a dominant trait and may occur in Down's or Hurler's syndrome.

**Heberden's nodes** (Fig 7 17). Bony nodules or outgrowths at the terminal interphalangeal joints, commoner in women and frequently associated with osteoarthritis of joints. They may occur in perfectly normal individuals.

**Bouchard's nodes.** Nodes on proximal inter-phalangeal joints in osteoarthritis.

**Haygarth's nodes.** Warm, pink and tender joint swellings involving the fingers, associated with arthritic changes.

**Osler's nodes** (Fig 7 22A). Small, raised, painful, tender, bluish or pink, usually pea-sized cutaneous nodules over the pads of the fingers (or toes) and thenar and hypothenar eminences, of sudden onset and usually lasting for one to five days, may be seen in subacute bacterial endocarditis. They are usually recognized only by blanching the area with pressure from a glass slide. Once thought to be embolic in origin, the nodes are now considered to be the result of an allergic vasculitis. Osler's nodes have also been reported to occur in systemic lupus erythematosus, nonbacterial thrombotic endocarditis, and gonococcal and typhoid infections.

**Janeway lesions.** Small painless, sometimes nodular, erythematous or haemorrhagic lesions over the palms of the hands (or soles) in cases of bacterial endocarditis, usually of the acute variety.

**Calcinotic nodules.** These are not unlike small tophi and may be seen in hypercalcaemia and scleroderma.

**Red finger tips** (tufted erythema) may signify small or intermittent right-to-left shunts with only slight reduction in arterial oxygen saturation.

**Pale hands, pink tipped.** In SBE fingers may be pale and the tips may be flushed.

**"Blanched" fingers.** In Raynaud's syndrome, usually associated with vasomotor instability in women, after exposure to cold, the fingers become exsanguinated, deadly white or "blanched", cold, painful and insensitive for varying periods of time, the blood returning slowly to the affected parts.

**Sclerodactyly.** Scleroderma or pathological thickening of the skin, when involving the fingers, may make them stiff and shiny or glistening, with a tendency to gangrene. This condition is frequently associated with Raynaud's disease.

**Trophic changes** Vascular insufficiency, such as occurs in atheroma, embolism, arteritis or cervical rib compression of the brachial artery leads to a skin change which may occur only at the finger tip. Lesions of the nervous system may give rise to mild trophic changes, or severe changes with ulceration and trauma-induced lesions. An increasingly common atrophic lesion in drug addicts results from an end-arteritis following intra-arterially injected drugs.

**Thyroid acropachy** This resembles clubbing of the fingers but the subperiosteal new bone formation with which it is associated is more often patchy than linear in distribution.

**Gangrenous fingers** Gangrene of one or more fingers of one or both hands may be due to vascular causes (e.g. Raynaud's syndrome, (Fig. 7 18) or thromboangitis obliterans (Fig 7 19). Loss of fingers may result from gangrene or leprosy.

**Morran's disease** A rare variety of trophic disease, probably secondary to syringomyelia or myelodysplasia, associated with painless whitlows on the fingers of both hands.

## THE NAILS

The nails of the fingers (and toes) may become involved in a variety of dermatological and systemic disorders. The following are some of the most important affections of the nails.

**Congenital abnormalities** These are extremely rare and may take the form of anonychia (complete absence of nails), micro- or macronychia, or onychoheterotopia (abnormally situated nail e.g. on the volar aspect of the finger), racket nail a congenital abnormality of the thumb with the nail conforming to the altered shape of the thumb, leuconychia totalis, periodic shedding of nails, excessive pitting or ridging of nails.

**Fungus infections** (*Tinea unguium*) (Fig 7 20) Infection with dermatophyte fungi such as *Trichophyton rubrum* is probably the commonest nail disorder. The fungus enters the nail bed distally and may cause only brownish-yellow discolouration adjacent to the point of entry. Spread towards the nail root leads to softening, fragility and irregular thickening of the nail with areas of onycholysis (separation of the nail from the nail bed).

**Chronic paronychia** Loss of cuticle damaged by constant wetting or at times manicuring allows a variety of bacteria and *Candida albicans* to enter the potential space between the nailfold and the underlying nail. It is mostly seen in housewives and domestic workers, though thumb-sucking children may develop similar changes. Chronic painful swelling occurs around the nail particularly of the right index and middle fingers and there is an intermittent purulent discharge in acute paronychia.

**Involvement in skin diseases** Nail changes may be the presenting, or at times an early manifestation of psoriasis, lichen planus, alopecia areata and

**vitiligo** In psoriasis the nail changes are a combination of localised areas of onycholysis, pitting of nails or thickening underlying the nail. In lichen planus pronounced longitudinal ridges may form and occasionally there is permanent atrophy and scarring. Pitting of the nails is common in alopecia areata and similar changes may occur in vitiligo.

### **Involvement in systemic disease**

*Impaired peripheral circulation* Chronic arterial insufficiency of whatever cause leads to excessive longitudinal ridging and thinning of nails (dystrophy of nails), and when severe may cause atrophy and scarring.

*Beau's lines* (Fig 7.21) Any severe illness physical or mental may cause transverse ridges to appear in the nail bed. These ridges or lines begin at the lunula and move towards the periphery as the nail grows outward. Since it takes approximately three months for a nail to grow out completely the date of onset of the illness may be roughly estimated by observation of the location of the furrow and noting its distance from the lunula. When lines appear on isolated nails the cause is likely to be local e.g. Raynaud's syndrome or in association with carpal tunnel syndrome.

*Mee's lines* These are transverse bands in the nails and may occur in Hodgkin's disease, high fevers, local nutritional derangement or chronic arsenic poisoning.

*Onycholysis* Onycholysis is often seen in thyrotoxicosis and less often in hypothyroidism.

*Koilonychia* (Spoon nails) (Fig 7.22) Concave, hollow or saucer-shaped nails, usually thin and with edges raised and centres depressed are characteristic of iron deficiency anaemia but may be seen transiently in normal babies and in manual workers constantly doing oily work. They have also been observed in thyrotoxicosis and rheumatic fever.

*Splinter hemorrhages* (Splinter fingers) (Fig 7.22A) Linear hemorrhages or hemorrhagic streaks under the nails are of particular importance in the diagnosis of subacute infective endocarditis. However, they may be secondary to trauma, trichinosis, rheumatic fever, infectious mononucleosis, cryoglobulinaemia, severe hypertension, severe rheumatoid arthritis, widespread malignancy, or may exist without an obvious cause.

*Abnormal shape* Broad or square nails occur in acromegaly and cretinism. Long and narrow nails may be observed in hypopituitarism and eunuchoidism. Parrot beak nails are seen with finger clubbing.

### **Colour changes**

*Black nails* occur in pseudomonas infection.

*Brown nails* may be seen in chronic renal disease.



*Yellow nails* occur in yellow nail syndrome of increased transverse curvature, slow rate of growth and increasing yellow discolouration of nails seen mostly in old men and may be associated with lymphatic oedema, idiopathic pleural effusion, chronic chest infection and myxoedema. Prolonged tetracycline ingestion may also cause yellowish discoloration of nails.

*White nails* with normal texture (leuconychia) are of four major types: total, partial, striate and punctate. All but the punctate form may be hereditary and all four forms may be acquired.

The total and partial forms usually follow a severe longstanding systemic disorder or local insult. In the total form there is loss of the lunula and the nail is opaque white. This is most commonly seen in hepatic cirrhosis but is also reported in typhoid fever, exposure to extreme cold, ulcerative colitis and trauma from nail-biting.

In the partial form the proximal nail is white and the distal nail has a transverse band of normal pink colouring. This is associated with hepatic cirrhosis, metastatic carcinoma, leprosy, exposure to severe cold, and nail-biting.

*Leuconychia striata* may follow a short or recurrent systemic disorder or local trauma, and presents as one or more complete transverse lines, of similar colour and parallel with the lunula. The lines are uniform 1-2 mm in width with smooth edges. This is seen in severe chronic hypoalbuminaemia due to chronic renal disease, myocardial infarction, cardiac insufficiency, pneumonia, Hodgkin's disease (in which they indicate a bad prognosis), chronic arsenic poisoning and local trauma resulting from manicure.

*Leuconychia punctata*, white patches on the nails, is very common and has no clinical significance. It often results from minor trauma to the nail matrix.

*White bands* In hypoalbuminaemia curved, narrow white bands may be observed parallel with the lunula (Muchrecke bands).

*Blue nails* may result from administration of mepacrine.

*Blue or azure half-moons* In Wilson's disease the lunulae may show bluish discolouration from deposition of copper.

*Red half-moons* may be seen in congestive cardiac failure.

*Half-and-half nail* This is a descriptive term given to the nail in which the proximal half is pale and the distal half is red, pink, or brown with a sharply demarcated line between the two halves. It is seen in azotaemia when the creatinine clearance is markedly diminished.

*Multiple pigment spots* may occur in Addison's disease.

## Tumours

*Warts* around the finger nails may occur in nail biters.

*Subungual exostoses* These present as hard nodules, usually on the toes, which distort the nail.

*Subungual fibromata* Warty swellings arising from the lateral edges of the nail base may be seen in tuberous sclerosis (epiloia)

*Glomus tumor* The nail bed is rich in neurovascular glomus bodies which very occasionally undergo benign enlargement to produce the exquisitely painful glomus tumor sometimes seen through the nail as a small dark spot

*Malignant melanoma* Melanoma may occur in the nail bed either as a pigmented lesion masquerading as a non-pigmented friable mass which may destroy the nail

### Miscellaneous conditions

*Brittle nails* These may be due to systemic causes such as impaired peripheral circulation or iron deficiency anaemia, or local cause such as constant immersion in water especially if alkaline, or use of nail varnish or cuticle removers

*Capillary pulsations* Pulsations of the capillaries in the nail bed can be observed in aortic regurgitation and in hyperdynamic circulatory states, and are best illustrated by exerting slight pressure over the distal end of the nail

*Hypertrophied nails* (onychauxis) are more commonly associated with the toes and may develop into massive 'hornlike' structures particularly on the big toe (onychogryphosis)

In *hereditary haemorrhagic telangiectasia* there are, particularly in the nail folds or beneath the nails, small violaceous blood-filled capillaries and arterioles which blanch on pressure

*Median canaliform dystrophy* This curious dystrophy of unknown etiology usually affects only one nail, typically the thumb. The deformity consists of a central longitudinal split with bilateral depressions extending outwards from this giving an 'inverted fir-tree' appearance. The condition clears spontaneously

*Egg-shell nails* have been described in avitaminosis A

## THE LOWER EXTREMITIES

### DEFORMITIES

Deformities of the lower extremities which may be either congenital or acquired are both common and important. They may result at times in other deformities, disabilities or abnormalities of gait.

*Congenital dislocation of hip* This may be unilateral with shortening of one leg, curvature of spine and a painless limp or bilateral with marked lordosis, prominent abdomen and buttocks and a waddling gait.

*Coxa vara and coxa valga* These are two opposite conditions, with either an abnormally small (less than 120 degrees) or large (over 140 degrees) angle between the neck and shaft of the femur

**Genu varum (Bow leg)** An outward bowing of the legs with the knees wide apart resulting from rickets (Fig 7 23), osteomalacia, osteitis deformans or achondroplasia

**Genu valgum (Knock-knee)** The femora being directed further inward than normal, bring the knees together and the feet apart (Fig 7 26) The condition may be either congenital or due to rickets or chronic ill-health

The term *genu recurvatum* refers to hyperextension of the knee-joint and *genu extrorsum* to valgum deformity of one knee coupled with varum deformity of the other

**Feet in acromegaly** The bones of the feet (and hands) are markedly enlarged in acromegaly (Fig. 7 25) The skin and subcutaneous tissues are thickened and enhance the bulk of the extremities

**Bowing of tibia** Bilateral symmetrical bowing of the tibiae, which is fairly common, may be due to rickets (usually lateral bowing of normal-sized tibiae in their lower halves), congenital syphilis ("sabre tibiae", with thickened and rounded borders and antero-posterior bowing of the entire bones) (Fig 7 26), osteitis deformans, osteomalacia or old multiple fractures

**Talipes equinovarus (Club foot)** (Fig 7 27) A common deformity of the foot of congenital origin but sometimes secondary to neurological disease, with both feet adducted downwards and inwards with a medial concavity Both soles face inwards on walking, the weight of the body resting on the outer borders The other varieties of talipes (viz. equinus, varus, valgus and calcaneus) are much less common

**Pes planus (Flat foot)** A common and bilateral, frequently congenital deformity of the foot, caused by a loss of the longitudinal arch of the foot and resulting in adduction and eversion of the foot at the midtarsal joint

**Pes cavus (Claw foot, pes arcuatus)** Exaggeration of the longitudinal arch of the foot resulting in a marked upward convexity of the instep and drawing up of the toes This deformity of the foot is usually bilateral Some are idiopathic but often familial Peroneal muscular atrophy is commonly associated with pes cavus as also Friedreich's ataxia (Friedreich's foot) It may be seen with spina bifida occulta or in those suffering from various forms of myelodysplasia Pes cavus is also seen in syringomyelia Anterior poliomyelitis may leave pes cavus in its wake because of muscle wasting and weakness in legs and feet Chronic muscular wasting, as in muscular dystrophy sometimes leads to pes cavus A spastic child may have pes cavus as well as other deformities

**Hallux valgus** A common bilateral deformity of women wearing high-heeled or pointed shoes, characterized by a lateral bending of the great toe at the metatarsophalangeal joint, with huddling together of the other toes The medial surface of the head of the first metatarsal soon develops an adventitious bursa and an area of thickened skin (bunion)



Fig 7.23 Bow legs  
(genu varum)

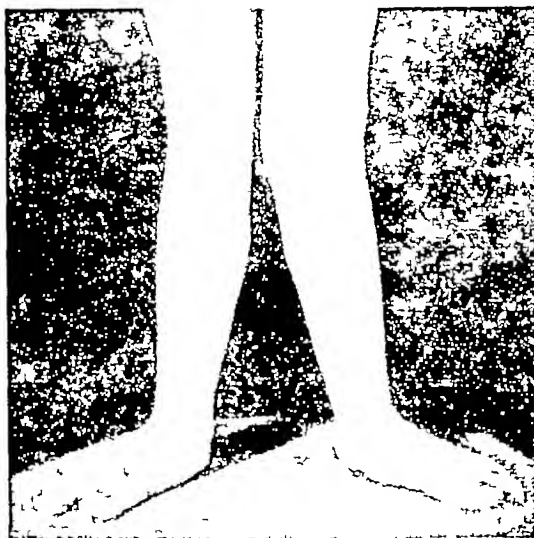


Fig 7.24 Knock knees (genu valgum)



Fig 7.25 Acromegalic feet compared with normal

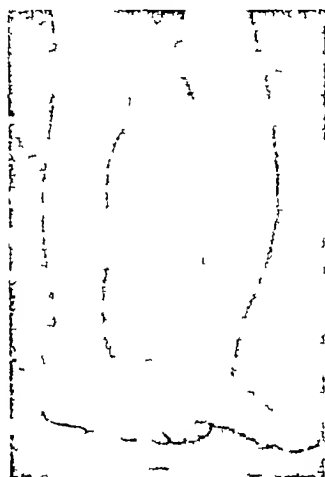


Fig 7.26 Bowing of tibia (saber tibia)



Fig 7.27 Club feet  
(Talipes equinovarus)

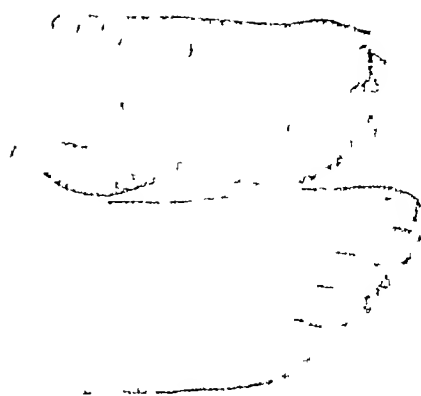


Fig. 7.30 Gangrene of the foot

OF O  
FACILITY

ENTRE

Fig. 7.29 Lym  
(elephant)

Fig. 7.31 Aneurysm  
external iliac artery

Fig. 7.22

**Congenital anomalies** Rare congenital malformations (Fig 7 28) similar to those of the upper extremity may also arise in the case of the lower extremities, e g complete or partial absence of the leg, extra toes, fused or webbed toes, extra phalanges, etc Fusion of the legs (sympodia), resulting in a mermaid type of individual has also been described

#### LOCAL SKIN CONDITIONS

Although the great majority of skin lesions appearing on the lower extremities are no different from and frequently a part of those appearing elsewhere, certain skin disorders, which show a special affinity for the lower extremities, require consideration

**Erythema nodosum** It is characterized by painful, tender, oval, hard and red swellings, about 1 to 2 inches in diameter, usually on the front of the legs which disappear after numerous changes in colour within seven to ten days It may be an important cutaneous sign of systemic disease It is supposed to be a hypersensitivity phenomenon Recognised associations include primary tuberculosis, sarcoidosis, staphylococcal pharyngitis, coccidioidomycosis, drug hypersensitivity, leprosy, and ulcerative colitis An association between erythema nodosum and brucellosis has also been reported

**Varicose eczema and varicose ulcers** A congested and purplish patch of skin, on the inner or outer surface of the leg in middle-aged or elderly subjects, later becoming chronically eczematous and scratchy, is a common finding in cases of varicose veins In a few of these cases, a typical varicose ulcer of variable size develops with overhanging and indurated edges, round or oval outlines, and a moist and red base

**Bazin's disease (Erythema induratum scrofulosorum)** Tender, purplish subcutaneous nodules of tuberculous origin, on the back and sides of the lower halves of the legs in young women, commoner in the winter months and with a tendency to resolution or formation of chronic and punched-out ulcers

**Erythrocyanosis crurum** A cold bluish-red swollen area, 3 or 4 inches wide, on the outer aspect of the lower third of the leg in young women, causing a persistent and unsightly swelling about the ankle

**Livedo reticularis (Marble skin)** A skin condition characterized by small white patches separated by a network of bluish-red lines, on the legs of children exposed to cold

**Thrombophlebitic ulcer** A thrombophlebitic ulcer usually follows an attack of thrombophlebitis of the legs after years and occurs invariably on one or other side of the lower third of the leg Chronic, painful and usually surrounded by a patch of dermatitis or skin pigmentation, the thrombophlebitic ulcer is variable in size and shape, and has usually a granular and dirty base

**Pre-tibial myxoedema** This is termed pre-tibial because of its distribution The raised, non-tender, thickened, bluish-red infiltration of the skin in hyperthyroidism is often easier to feel than to see

**Perforating ulcer** So called because of its deep extension, the perforating ulcer is usually a trophic lesion situated under the ball of the great toe or the outer edge of the sole Starting as a corn-like hypertrophy, it soon forms a

deep, painful, indolent, foul-smelling, soft, discharging ulcer, intractable to treatment. It is usually found in *tabes dorsalis* and diabetes but may occur in Raynaud's syndrome, Buerger's disease, spastic paralysis or leprosy.

**Syphilitic ulcers** Secondary to subcutaneous gummata, syphilitic ulcers are usually multiple, circular punched-out and with a tendency to confluence.

**Athlete's foot.** Ringworm (usually epidermophyton or trichophyton) infection of the clefts between the toes, resulting in patches of dry dead skin during remissions, and redness, swelling and oozing of the skin during acute phases.

## O E D E M A

Oedema or excessive accumulation of fluid in the subcutaneous tissues is extremely common in the lower extremities. It may be part of a generalized oedema, or confined to one or both legs, or to small sections of the leg.

*Early oedema*, frequently obvious on "pitting", may be detectable in the region of the ankles (especially in ambulant cases), or over the sacrum (sacral pad), trochanters (trochanteric pads) or back of thighs (especially in recumbent patients) or in the region of the genital organs (particularly the scrotum)

Oedema of the legs may be of *venous type*, soft and pitting oedema, with smooth, shiny, bluish and cold overlying skin and no evidence of regional lymphadenopathy as in congestive cardiac failure or cirrhosis of the liver, of *lymphatic type* (Fig. 7.29), much less common, solid and non-pitting form of oedema, with thickened, coarse, pitted and warm overlying skin and frequent association of enlarged regional lymphnodes as in filariasis or malignancy of lymphnodes, or rarely of *myxoedematous type*, localized, non-pitting, puffy swellings over the legs in hypothyroidism

Oedema of the legs may be bilateral or unilateral. *Bilateral oedema* may be physiological as in pregnancy, obesity or hot weather, or pathological and due to congestive cardiac failure, nephritis or nephrosis, hypoproteinaemia, vitamin B<sub>1</sub> deficiency (wet beri-beri), epidemic dropsy, intra-abdominal causes (such as cirrhosis of the liver, ascites or malignancy, impeding flow of blood through the inferior vena caval system), bilateral affections of leg veins (e.g. thrombophlebitis or varicose veins) or lymphatic obstruction (filarial or non-filarial) or to local causes (such as tight garters or socks)

*Unilateral oedema*, which is less common, may be due to unilateral affections of leg veins (e.g. varicose veins, thrombophlebitis or phlebothrombosis), lymphatic obstruction (filarial or non-filarial elephantiasis), local inflammation, angioneurotic oedema, neurological conditions (e.g. hemiplegia or monoplegia), neurofibromatosis (elephantiasis neuromatosa), pregnancy (phlegmasia alba dolens) and Milroy's disease ("chronic hereditary oedema", a rare, familial and persistent form of oedema)

## LYMPHNODES

During examination of the lower extremities, three groups of lymphnodes must be examined for evidences of enlargement or disease, viz the superficial inguinal group in the subcutaneous tissues near Poupart's ligament, the femoral and deep inguinal group below the saphenous opening, and the popliteal group in the popliteal fossa

*Inguinal lymphadenopathy*, involving the superficial and deep inguinal groups, may be due to septic infection (anywhere on the lower extremity or perineal region), epithelioma or carcinoma of the scrotum, penis, perineum, or lymphogranuloma inguinale, bubonic plague, rubella, filariasis, lymphadenoma, lymphatic leukaemia, pediculosis pubis, or even excessive physical strain

*Popliteal lymphadenopathy*, usually mild and difficult of detection, may be due to septic infection anywhere over the foot or leg

## JOINTS

See examination of the arthritic patient

## BONES

The various types of abnormalities of the bones of the lower extremities, viz, bony tenderness, deformities, curvatures, local swellings and tumours, are no different from those of the upper extremities

## ARTERIES

**Occlusive arterial disease** In the lower extremities, peripheral vascular disturbances, with serious consequences like gangrene (Fig 7 30) and ulceration, are particularly common and may result from arteriosclerosis obliterans, thromboangitis obliterans, or rarely from vasospastic or vasomotor syndromes (such as Raynaud's syndrome) Intermittent claudication, severe nocturnal rest pains or subjective feelings of coldness or numbness associated with "colour" changes, trophic disturbances, objective coldness of the foot, and complete or partial obliteration of arterial pulsations in the palpable arteries of the legs (dorsalis pedis, posterior tibial, popliteal and femoral arteries), collectively constitute a clinical picture highly suggestive, if not diagnostic, of occlusive arterial disease This is followed in many cases by subsequent gangrene or ulceration The nature of the causative disease is usually suggested by the age, sex and clinical features and confirmed by special investigations

**Aneurysms** in the lower extremities are not uncommon and may involve the external iliac (Fig 7 31), femoral (Fig 7 32) or popliteal artery They are usually traumatic in origin



## VEINS

**Varicose veins** An extremely common condition, characterized by prominent, dilated and tortuous superficial veins in one or both lower extremities (Fig 7 33), commoner in women, sometimes familial and liable to complications like thrombophlebitis, eczema and ulceration (varicose ulcers)

**Venous thrombosis** In majority of patients, the thrombosis occurs in the veins of the calves of the legs. The thrombosis is usually non-inflammatory (*phlebothrombosis*) and clinically silent. Presence of inflammation (*thrombophlebitis*) is generally associated with pain. The diagnosis of *superficial thrombophlebitis* is usually straightforward. The patient complains of pain and swelling along a superficial vein which is reddened and tender to touch. Fever is often present. This type of lesion rarely has complications but one must be on the lookout for underlying malignancy, especially in a case of recurrent thrombophlebitis.

**Deep vein thrombosis** may or may not have symptoms or signs. Early signs include warmth in the affected leg and tenderness of deep calf veins best elicited by keeping the calf muscles relaxed (by flexing the knees) and working the fingers up slowly from the heel to the popliteal space. Dorsiflexion of the foot may cause pain in the calf (Homan's sign). Slight distension of the veins may be noticed on the dorsum of the foot on the affected side. Swelling of the leg and discolouration are late signs. Clinical conditions associated with an increased risk of venous thrombosis and pulmonary embolism include prolonged bed rest particularly with congestive cardiac failure, trauma especially lower limb fractures, surgery especially abdominal and pelvic, and post-partum.

## VASOMOTOR SYNDROME

**Raynaud's syndrome or disease** may affect either the upper or lower extremities or both, mostly in young women. The affected toes or fingers go through characteristic cycles of pallor (blanching), cyanosis and rubor, on exposure to cold.

**Erythromelalgia** A vasomotor disturbance of the extremities associated with patent arteries, severe burning and redness and aggravated by standing, exertion or the application of heat.

**Acrocyanosis (Acroasphyxia)** A persistent and symmetrical purplish discoloration of the feet, ankles (or hands) associated with coldness and sweating, in young women.

## MISCELLANEOUS CONDITIONS

**Poor muscular development** In coarctation of the aorta, the lower limbs may show poor muscular development in contrast to the upper extremities.

**Anterior tibial syndrome** A characteristic syndrome of pain, swelling, and ischaemic necrosis of the anterior tibial group of leg muscles, resulting in foot-drop, it may result from trauma or undue strain and may be transitory or persistent.

**Anxietas tibiarum** ("Jittery leg syndrome") Severe paraesthesiae or pains in the legs, resulting in restless movements of the legs at rest or during sleep, and frequently lasting for years. It has been variously regarded as vasomotor, functional or organic nervous disorder.

**Lipedema** (Fat-legs) Progressive, slow, tender and painful enlargements of both legs in women, more marked on standing and in hot weather, frequently accompanied by feelings of depression or frustration.

**Metatarsalgia** Acute and severe burning pain on walking, in the fourth (or second) toe of the foot, with a tendency to progression, frequently caused by faulty footwear.

**Local warmth** An arterio-venous aneurysm, congenital or due to Paget's disease of bone may cause local warmth.

**"Painful heel"** Severe pain or tenderness in the region of the heel, a common and intractable symptom may be due to a "calcaneal spur" (over the tuberosity of the os calcis), calcaneal apophysitis (degeneration of the epiphysis), a partial separation of the epiphysis, a retro-calcaneal bursitis or tenosynovitis of the tendo Achilles.

**Ingrowing toe nail** (Onychocryptosis) An extremely common and painful condition of inflammation of the soft tissues at the side or corner of the nail of the great toe, with associated redness, swelling and tenderness.

**Onychogryphosis** (Club nail) A massive, "horn-like" hypertrophy of the nail of the great toe in elderly subjects, resulting in considerable inconvenience and disfigurement.

## EXAMINATION OF THE ARTHRITIC PATIENT

An accurate diagnosis is essential in a patient with joint disease. Just as one looks for a cause of fever, one should also look for the cause of arthritis since neither of these terms is a diagnosis.

### HISTORY

A detailed history including preceding illness, mode of onset and rapidity of progress, severity, location of swelling, precipitating or aggravating factors, history of previous injury or trauma, and treatment if any in the past is essential.

The *family history* may suggest gout, rheumatoid arthritis or Heberden's nodes.

### PHYSICAL EXAMINATION

A complete physical examination should consist of the following  
**General examination**

General appearance including gait and posture. Skin lesions suggestive of psoriasis, lupus erythematosus, erythema nodosum, scleroderma or purpura.

Cold moist extremities are suggestive of rheumatoid arthritis, markedly hot of infectious arthritis or gout.

Subcutaneous swellings, e.g. nodules in elbow area in rheumatoid arthritis or subcutaneous tophi of gout (Fig. 7.4).

Cardiac murmurs in rheumatic fever, ankylosing spondylitis or syphilis

Lymphadenopathy, splenomegaly, hepatomegaly in juvenile rheumatoid arthritis, or in lupus erythematosus

Moon face, striae, acne, etc as evidence of steroid therapy

### Local examination of joint

It is essential that the part to be examined should be adequately exposed to a good light.

### Inspection

*Overlying skin* Colour (redness or pigmentation), shininess and amount of sweating

*Periarticular tissues* Diffuse oedema of subcutaneous tissue about the joint or localised joint swelling in adjacent bursae or tendon sheaths

*Bones* For deformity or unusual posture

*Scars and ulcers* Irregular in injury, puckered and adherent in suppuration or due to previous operation

### Palpation

*Skin temperature* Comparison of the two sides must be made for warmth usually due to inflammation or less commonly coldness as in rheumatoid arthritis

*Soft tissues* Synovial or capsular thickening is appreciated by a doughy feel in case of joint where the joint capsule and synovial affection are near the surface Effusion in joint can be identified by demonstrating floatation in the joint sac e.g., floating patella in case of knee joint

*Bony enlargement* Heberden's nodes at terminal interphalangeal joints, or sometimes osteophytes if sufficiently large may be palpable at the articular margins in osteoarthritis Abnormal prominence or disturbed relationship of normal anatomical landmarks should be noted

*Tenderness* The site and degree of tenderness should be noted

*Movements* (a) Range of active movement—Limitation of movements in different directions is suggestive of some type of arthritis Limitation may be due to trauma, effusion into joint, muscle spasm, contracture of periarticular tissues, fibrosis or bony ankylosis (b) Crepitation on motion Coarse crepitus is common in the knees in degenerative joint disease

*Muscle changes* Wasting, spasm, weakness or shortening in the flexor muscles of a diseased joint

*Nodules* Rheumatoid nodules adjacent to finger, elbow and knee joints, gouty tophi Loose bodies (joint mice) can be felt at times in the knee, ganglia and synovial cysts especially at the wrist

*Measurements* To detect muscle wasting, soft tissue swelling or bony changes, measurement of the limb segment on either side is useful

### Classification of arthritis

- 1 Rheumatoid arthritis and its variants, such as psoriatic arthritis, juvenile rheumatoid arthritis, Reiter's syndrome Ankylosing spondylitis is now accepted as a separate disease entity because of its different clinical course and histology
- 2 Rheumatic fever
- 3 Degenerative joint disease (osteoarthritis)
- 4 Arthritis associated with known infections e.g. tuberculous, gonococcal, staphylococcal, pneumococcal, syphilitic, viral arthritis, etc
- 5 Neuropathic arthritis (Charcot's joint)
- 6 Arthritis associated with known biochemical or endocrine abnormalities Gout, hyperparathyroidism, ochronosis, acromegaly, hypothyroidism, scurvy
- 7 Traumatic
- 8 Allergy and drug reactions
- 9 Blood disorders Haemophilia, haemoglobinopathies (sickle cell disease), agammaglobulinaemia
- 10 Connective tissue disorders
- 11 Tumors and tumor-like conditions Benign tumors, metastatic tumors, multiple myeloma

### Acute arthritis

*Rheumatic fever* While usually affecting school children it can occur in adolescents and adults. It may start with one joint but polyarthritis usually develops and is characteristically flitting, involving large joints and rarely remaining in any one joint for more than a few days. In adolescents, the small joints may be involved and symptoms and signs may last longer. The typical rash, erythema marginatum, occurs only in a small proportion of cases and so do rheumatic nodules. Cardiac involvement is common.

*Septic arthritis* (Fig 7 34) In this form of arthritis, the joint is infected by bacteria of one of the pyogenic groups. There are local signs of acute inflammation in one or more joints associated with fever and rigors.

*Viral arthritis* An acute benign arthritis may precede, coincide with or follow a variety of viral infections. Usually affecting older children and adolescents, it may involve any joint, is short lived and resolves completely. Most commonly seen in rubella, and after immunisation for rubella, it can occur in mumps, glandular fever and infectious hepatitis.

*Traumatic arthritis* The history of the onset helps in making a diagnosis, but minor trauma may precipitate acute lesions like gout, bursitis of shoulder or haemarthrosis especially in patients with blood dyscrasias (Fig 7 35).

*Still's disease* It affects more girls than boys and the presentation varies from a serious systemic illness with fever, to a single swollen and often painless joint. Swinging fever with arthralgia or arthritis, maculopapular rash are helpful in diagnosis, as also the occasional presence of lymphadenopathy, splenomegaly and hepatomegaly.

*Gout* Acute gout is more common in males. Classically it starts in the great toe (Fig. 7 36), although any joint can be the first to be affected. The affected joint is swollen, red, shiny and extremely painful. Serum uric acid is raised.

*Pseudo-gout* Affecting both sexes equally, it presents with an acute arthritis but rarely involves the great toe, the knees being one of the commonest sites. An absolute diagnosis depends on the demonstration of calcium pyrophosphate crystals in the synovial fluid.

*Henoch-Schonlein purpura* There is a peak age of onset between two and five years, while rheumatic fever is rare in the preschool child. The characteristic rash is a maculopapular eruption with purpura on the buttocks and extensor surfaces of the limbs particularly the legs, and is associated with an acute arthritis, predominantly of large joint and often migratory. The joint symptoms usually subside quickly and without residual damage.

*Allergic (Serum sickness)* This occurs 2-10 days after injection of serum and is characterised by arthralgia or arthritis of larger joints, urticarial or morbilliform rash and fever.

*Drug reactions* A number of drugs notably penicillin, some anticonvulsants, procainamide, hydralazine, anti-tuberculous drugs and griseofulvin may cause arthralgia and arthritis with rash and sometimes fever.

### Chronic arthritis

*Rheumatoid arthritis* The clinical features are extremely variable. Prodromal symptoms include vague pains in the muscles and a general deterioration of health. These are followed by a slow onset of polyarticular stiffness, pain and swelling involving the metacarpophalangeal joints and proximal interphalangeal joints of the fingers (Fig. 7 37), the wrists and metatarsophalangeal joints, and subsequently knees (Fig. 7 38), ankles, shoulder, elbows and other joints. In addition to joints many other tissues of the body may be affected and include rheumatoid nodules, nerve entrapment, inflammatory infiltration of lacrimal and salivary glands, eye lesions and systemic features such as anaemia, weight loss and fever. Occasionally involvement of a single large joint may occur.

*Psoriatic arthritis* There is fusiform swelling of distal interphalangeal joints of fingers and interphalangeal joints of the thumb. Apart from the rare arthritis mutilans, psoriatic arthritis tends to cause less pain and disability than rheumatoid arthritis.

Fig 7.34 Septic arthritis  
of left knee joint

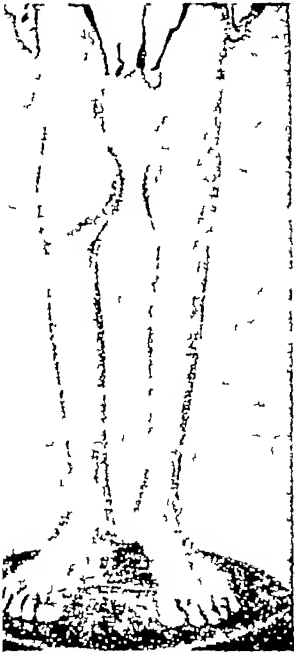


Fig 7.35 Haemophilic  
left knee joint. Note the  
ecchymoses on the  
medial aspect of the left  
knee

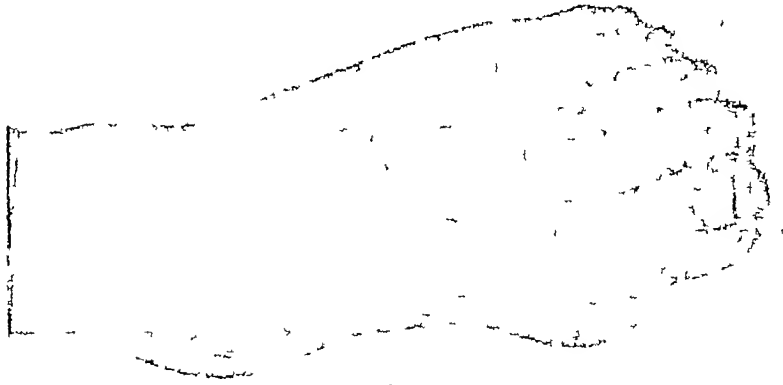


Fig 7.36 Acute gouty arthritis involving big toe



Fig. 7.37 Fusiform swelling of fingers Early rheumatoid arthritis

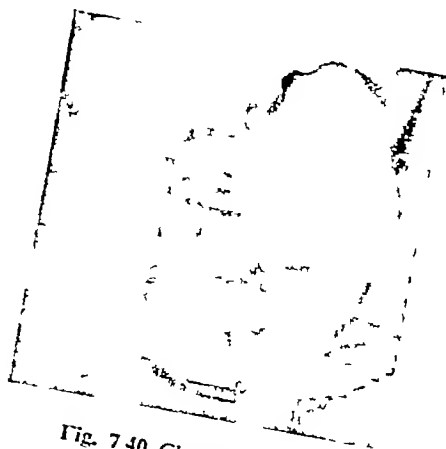


Fig. 7.40 Charcot's joint



Fig. 7.39 Congenital hip dislocation

of Note

*Feltz's syndrome* In addition to rheumatoid arthritis lymphnode enlargement may be found, with moderate enlargement of spleen, leucopaenia and sometimes leg ulcers

*Sjogren's syndrome* Rheumatoid arthritis is associated with keratoconjunctivitis sicca, dry mouth and enlarged salivary glands

*Reiter's syndrome* This disease is a triad of urethritis, conjunctivitis and arthritis, and may follow diarrhoea (post dysenteric form) or be venereally-acquired urethritis (post-sexual form)

*Ankylosing spondylitis* It is a disease of adolescence and has a familial incidence. Usually it begins in the sacroiliac joints and spreads to the spine to affect the costovertebral joints, the peripheral joints such as hips and knees being involved only occasionally

*Osteoarthritis* Usually affects elderly, often obese individuals. Joints most frequently involved are the knees, hips and carpometacarpal joints of the thumbs. Less commonly the shoulder, ankle and wrist joints are affected. Effusion and soft tissue swelling are uncommon except in the knees

*Tuberculous arthritis* Joints most often involved are large joints such as hip and knee, and the synovial joints between the vertebral bodies. Characteristic features include increased warmth of the overlying skin, swelling and limitation of movement in all directions. There is wasting of muscles controlling the joint. Abscess formation or formation of a sinus is common

*Syphilitic arthritis* Bone and joint involvement occurs in both congenital and acquired syphilis

*Clutton's joints* Symmetrical syphilitic synovitis usually occurring at the time of puberty and affecting mostly both the knee joints (Fig 7 39). The joints show painless enlargement, instability, thickening of synovial membrane and effusive Charcot's joint (Fig 7 40). Neuropathic arthritis which can occur in tabes dorsalis, syringomyelia, diabetic neuropathy, leprosy neuropathy and myelomeningocele. Also with other conditions causing sensory impairment in the limbs such as congenital insensitivity to pain, myelopathy of pernicious anaemia, spinal cord trauma, hereditary sensory neuropathy, Charcot-Marie-Tooth disease, and pyogenic arthropathy following intra-articular injections of corticosteroids



## 8

# Breasts, Back and Genitals

## THE BREASTS

EXAMINATION of the breasts should be routinely undertaken, during the course of physical examination, especially in female patients, as it may serve to disclose the presence of early and operable carcinoma, the commonest form of carcinoma in women of all ages

**Method** Examination of the breasts should be carried out, carefully and systematically, both by inspection and palpation and with the patient in the upright and recumbent positions

INSPECTION is usually carried out in the standing or sitting patient, first with the arms hanging by the sides, then with the arms raised above the head and finally with the patient bending forwards. The following features should be particularly noted (1) *Asymmetry of size or shape* of the breasts, undue largeness or smallness of one breast, besides being due to local disease, may be congenital (Fig 8 1) or caused by systemic diseases such as pulmonary tuberculosis (2) *Abnormalities of contour*, such as local bulging, retraction or dimpling, retractive phenomena are very suggestive of malignancy (3) *Colour and appearance of the skin*. Local redness of the skin may be due to acute mastitis, breast abscess or inflammatory carcinoma, "orange peel" (peau d'orange) appearance of the skin is almost diagnostic of malignancy (4) *Abnormalities of the nipples*, such as asymmetry of position or the presence of retraction, flattening, inversion, bulging, deviation, fissuring or eczematous condition of one or both nipples, the colour and character of nipple discharge, when present, must be noted, a sanguinous discharge is suggestive of carcinoma or papilloma within a duct. Characteristically pigmentation of Addison's disease is associated with an increased melanin deposition in the areola of the nipple (5) *Abnormal fullness or swellings* of the axillary and supraclavicular regions. These may be indicative of regional lymphnode enlargements, as in cases of breast carcinoma (6) *Congenital malformations*, such as accessory nipples or supernumerary breasts particularly along the "milk lines" from the axillae to the inner surfaces of the thighs

PALPATION of the breasts is best carried out with the patient supine, and the shoulders raised with a pillow. Each breast is palpated gently against the chest wall, with the flat of the fingers of one hand. The inner quadrants of the breasts are felt with the patient's arms raised above the head and the outer quadrants with the arms by the sides of the body. Each section of the breast is thus examined for the presence of a lump or lumps. The size, shape, surface margins, mobility, position, consistency and attachment to skin and adjacent structures of any such lumps, discovered by palpation, are carefully noted.

Retractive phenomena (such as "dimpling" or "indentation") over the breast are looked for particularly with the patient's arm raised or with the body bent forwards. They may also be revealed, at times, by manually lifting or compressing each breast.

In case of nipple discharge, a careful palpation of the superficial layers of the breast, particularly near the areolae, may disclose the origin of the discharge.

With the patient sitting opposite the examiner, the supraclavicular fossae and axillary regions are carefully palpated for evidence of enlarged or malignant lymph nodes. For a proper palpation of the axilla, relaxation of the pectoral muscle may be achieved by supporting the arm of the patient by the side of his or her body.

## DISEASES OF THE BREASTS

The breast, particularly the female breast, may be the seat of numerous affections or diseases, such as painful nodules, mastodynia, acute mastitis, breast abscess, fibroadenoma, chronic cystic mastitis, tuberculosis, syphilitic carcinoma, or sarcoma. Most of these are adequately dealt with in textbooks, of surgery. The following require special mention.

**Gynaecomastia** Enlargement of the male breast or gynaecomastia (Fig 8.2) is a frequent condition which must be differentiated from diffuse deposition of fat in the obese male. It may be unilateral or bilateral. It is important to examine the external genitalia to confirm that the patient has a normal penis or testicles, otherwise the diagnosis of hermaphroditism or pseudohermaphroditism may be missed. Gynaecomastia is seen in conditions which have little in common. (1) Physiological (a) Gynaecomastia of the adolescent (b) Klinefelter's syndrome (Fig 8.3) due to uninhibited secretion of pituitary breast stimulating hormone (c) Neonatal breast hypertrophy (2) Secondary to systemic disease (a) Hepatic disease Cirrhosis (b) Pulmonary disease Carcinoma of lung, bronchiectasis, tuberculosis (c) Severe malnutrition (d) Generalized skin disease (3) Pituitary syndromes Acromegaly, Cushing's syndrome, chromophobe tumours (4) Other endocrine diseases Hyper- and hypothyroidism, adrenal hyperplasia, diabetes mellitus (5) Testicular alterations Eunuchoidism, castration (6) Neurological syndromes Paraplegia, skull trauma, Parkinsonism (7) Iatrogenic Cyclopentaphenanthrene drugs Oestrogens, androgens, progesterone, desoxycorticosterone, spironolactones, anterior pituitary extracts and gonadotrophins, as well as amphetamine, digitalis, isoniazid and reserpine (8) Trauma to breast

**Carcinoma of breast** This is unquestionably the most important of all breast diseases. Its successful treatment depends on the early diagnosis, or detection, of a lump in the breast. Features suggestive of malignancy are hardness, nodularity, ill-defined margins, immobility, attachment to skin or deeper structures, "dimpling", indentation or retractive phenomena of the breast, flattening, inversion, asymmetry or deviation of a nipple, a sanguinous discharge, characteristic skin changes (such as a *peau d'orange* appearance), enlarged and hard lymphnodes in the axilla or supraclavicular region, and evidences of malignant deposits in the bones of the skull, pelvis or spine. It is important to note that in the early stages of breast carcinoma most of these features may be wanting.

**Chronic cystic mastitis** This is characterized by a diffuse "shottness" or nodularity of one or both breasts frequently associated with one or more large lumps or cysts, particularly in the outer quadrants of the breasts. The tense, elastic and large cysts or lumps of chronic mastitis are not only difficult to distinguish from carcinomatous lumps, but may become malignant in later years.

**Fibroadenoma of breast** This may take the form of one or more, smooth, firm, discrete, freely moveable and "slippery" lumps within the breast tissue in young girls or women. Although completely benign and unattached to skin and other structures, an occasional fibroadenoma may become sarcomatous later in life.

**Galactorrhoea** Persistent lactation due to excessive secretion of prolactin may result from (1) Hypothalamic lesions such as tuberculous meningitis (2) Pituitary lesions e.g., tumors, acromegaly, Cushing's disease (3) After chest trauma, thoracotomy or herpes zoster producing irritation of thoracic spinal nerve segments (4) Lung tumors (5) Drugs such as phenothiazines, reserpine, methyldopa, oral contraceptives and haloperidol.

## THE BACK

**Method** Examination of the back should be carried out in a good light, with the patient in various positions, both at rest (e.g. supine, prone, sitting, standing and stooping forwards or backwards) and in movement. The following features are particularly deserving of study, viz. standing posture, curvatures of spine, range of movement (both active and passive) of spine (cervical, thoracic and lumbar), local tenderness or muscle rigidity, deformities, local swellings (such as abscess, tumour, cyst or meningocele), skin lesions (such as bed sores), and affections of the scapulae.

**Spinal curvature** The *normal spine* displays a mild convexity (kyphosis) of the thoracic spine, a mild concavity of the cervical spine and a definite concavity (lordosis) of the lumbar spine, without any lateral curvature (scoliosis).

**KYPHOSIS** An abnormal kyphosis or convexity of the thoracic spine is common and may take one of two forms. A widely curved spine is suggestive of rheumatoid arthritis (spondylitis), general debility, Parkinsonism, Paget's disease, von Recklinghausen's disease, senile osteoporosis or growth disturbance of bones. A sharp angulation (so-called *gibbus*) is suggestive of Pott's disease (Fig 8 4), fractured spine or malignant disease.

**SCOLIOSIS** or lateral curvature of the spine (Fig 8 5) is important to recognize as it may lead to faulty diagnosis of heart or lung disease. Scoliosis may be either functional (in which case, it disappears on bending forwards), or

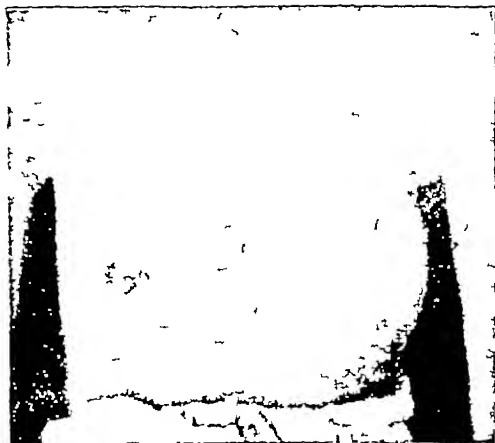


Fig 81 Congenital hemihypertrophy of left breast

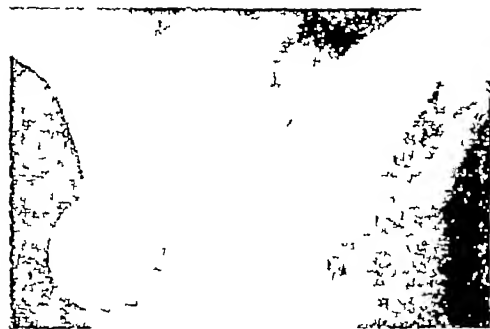


Fig. 82 Unilateral gynaecomastia

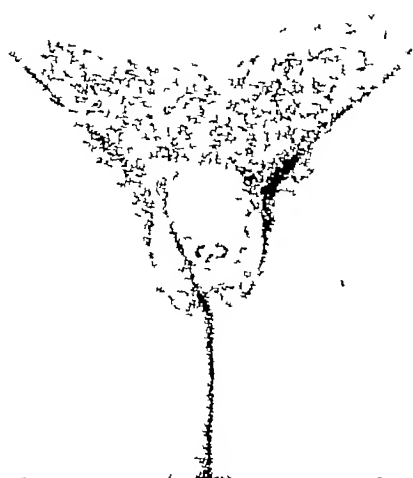


Fig 8.3 Klinefelter's syndrome  
Patient aged 22 years having  
absence of facial hair Note  
gynaecomastia and small external  
genitalia (Courtesy Dr P N  
Shah, Indian Cancer Research  
Centre, Bombay)

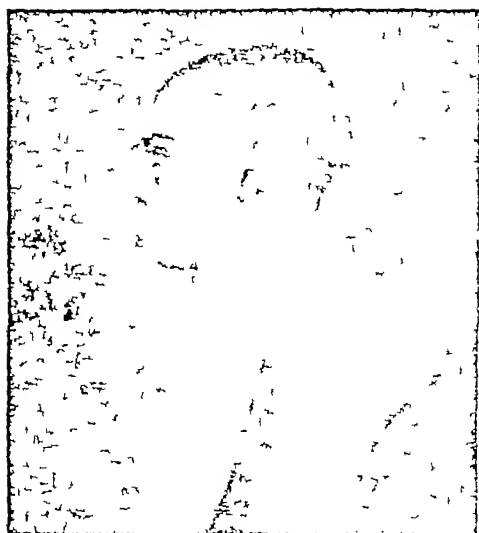


Fig. 84 Severe kyphosis with  
angular deformity

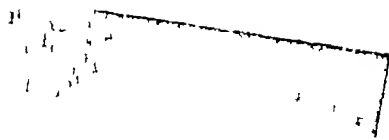


Fig 86 Lordosis of spine (hysterical)

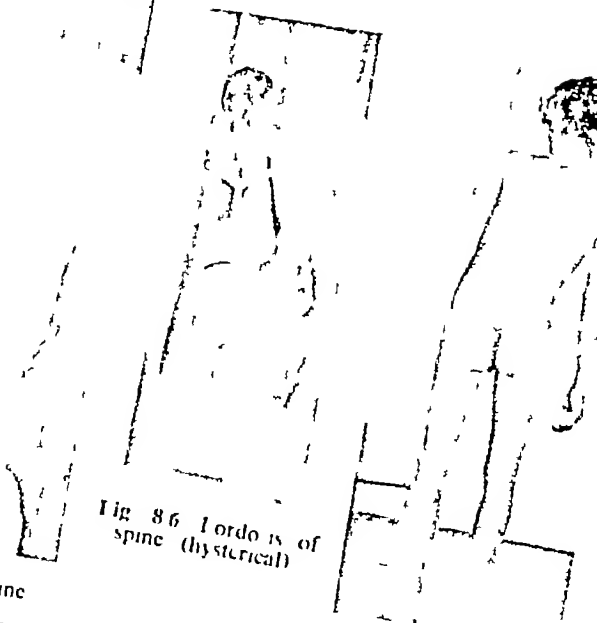


Fig. 87 Listing of the back  
Lateral bending towards the  
side of the list in a case of  
slipped intervertebral disc

to deformity of spine



to rules on  
back

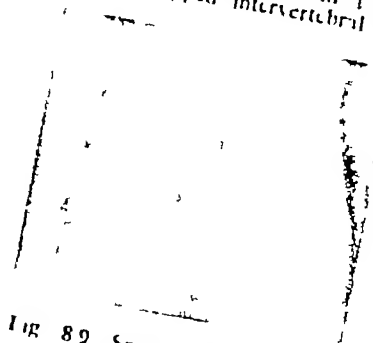


Fig 89 Spina bifida with a  
tuft of hair

Fig 810 Spina bifida  
meningocoele

structural, the curvature persisting during forward bending and associated with rotation of vertebral bodies. Structural scoliosis may be either congenital (with one or more hemivertebrae), idiopathic (of unknown cause, particularly in adolescent girls) or paralytic (secondary to poliomyelitis, rickets, etc.)

**LORDOSIS** An abnormal lordosis of the lumbar spine may be due to general debility, wasting disease, myopathy, poliomyelitis, faulty posture, pregnancy, abdominal swelling or hysteria (Fig 8 6)

**LIST OF THE BACK** (Fig 8 7) A laterally bent position of the spine may be assumed as a result of pain in ruptured intervertebral disc. A list of the back differs from scoliosis in that it is an acute condition and accompanied by pain.

**POKER SPINE** (Bamboo-spine) In ankylosing spondylitis, the spine is held rigid, being stiff and immobile with loss of normal curves.

**Swelling of back** A local swelling of the back may be easily visible if superficial, but, if deep-seated, it can only be detected after careful palpation, the patient being put in a prone position so that the spinal muscles are relaxed. Swellings of the back include tumours, meningocele, cold abscess, perinephric abscess, aneurysm of descending thoracic aorta and rarely appendicular abscess. Rheumatic nodules may occur along the spine (Fig 8 8).

**Spina bifida** This results from failure of the closure of the spinal column due to a defect in the development of vertebrae. The failure of closure may occur at any level, being most common in the lumbosacral region. The defect may be a simple failure (Fig 8 9), or portions of nervous tissue may protrude through the defect (Fig 8 10).

**Buffalo hump** In Cushing's syndrome there may be certain areas such as the dorsal cervical region with a propensity for fat deposition.

## THE GENITALS

### MALE GENITALIA

An examination of the male genital organs usually implies an inspection and palpation of the pubic hair, penis, scrotum, testicles, prostate and seminal vesicles. Special methods of investigation may prove necessary for examination of the urethra and prostate.

Involvement of male genitalia by disease may result in pain within the penis, testicles or rectum, discharge of blood, pus or mucus from the urethra, painful or precipitate micturition with frequency, priapism, or local redness, inflammation or swelling of the part affected.

**Local skin and hair.** The skin of the genital area and thighs may display evidences of intertrigo or tinea cruris (dermatophytosis) while the pubic hair may be affected by pediculosis cruris.

**The Penis URETHRITIS** Inflammation or redness of the glans penis and foreskin with a purulent urethral discharge and enlarged inguinal lymphnodes is highly suggestive of gonorrhoeal urethritis, a diagnosis confirmed by microscopic examination of the urethral discharge for gram-negative, intracellular diplococci. Much less common as causes of urethritis are trichomonas and other rare organisms, trauma, e g after catheterization and ingestion of irritant substances, such as cantharidis and excess of alcohol, and Reiter's syndrome (urethro-conjunctival-synovial syndrome)

**PHIMOSIS** The prepuce cannot be retracted or pushed back over the glans, because of an excessively small orifice or adhesions, a condition frequently congenital but at times secondary to inflammatory swelling.

**PARAPHIMOSIS** The prepuce remains retracted behind the corona and cannot be pushed forward, owing to inflammatory swelling. The condition may, at times, result in excessive swelling or gangrene of the part.

#### ULCERATIONS OF GENITAL REGIONS

Affections of the genitals may be in the form of ulcers or papillary warty growths. Genital ulcerations may be venereal or nonvenereal. The common causes of nonvenereal ulcers are pyogenic or traumatic ulcers, scabetic ulcers, herpes simplex, carcinoma or rarely other diseases of the skin or mucosa, primarily starting on the genitals. In the absence of a venereal history all the other causes should be considered.

**Venereal ulcers CHANCRE (HARD SORE)** In primary syphilis, a solitary superficial, non-tender, painless and indolent ulcer with a scanty serous exudate and induration of the base may appear over the glans penis or the under-surface of the prepuce. Rarely multiple, a hard chancre is usually associated with a painless, moderately large and hard swelling of the inguinal lymphnodes. The diagnosis is confirmed by dark-ground examination of the exudate for the presence of the causative organism, *treponema pallidum*.

**CHANCROID (SOFT SORE)** The sores are usually multiple, superficial or deep, rounded or irregular, have greyish-yellow bases, tend to bleed easily and discharge a lot of dirty yellow stuff. Surrounded by areas of redness, chancroids usually appear in persons with dirty habits, over or near the fraenum, and are accompanied at times by tender, suppurative enlargement of the inguinal nodes. In view of the frequent co-existence of syphilitic primary chancre and chancroids, a careful inspection of the genitals is essential in every case of "soft sores" in order to follow up for evidences of syphilitic infection by serological tests for syphilis after 8 to 12 weeks.

**GRANULOMA INGUINALE.** It is a venereal condition due to infection with *Bacillus Donovanii*. The ulcer has profuse granulomatous tissue on the floor and irregular turned out edges. It spreads by contiguity in the genitalia and groins. Lymphnodes are not involved unless there is a superadded secondary infection (Fig. 8 11)

**LYMPHOGRANULOMA INGUINALE** (Fig 8 12) A rare type of venereal infection in which a primary herpetic ulcer is seen (rarely) on the genitalia. Usually the patients come three to six months after the exposure with bilateral inguinal lymphadenitis and bubo formation. The glands break down with formation of multiple sinuses. This is a virus infection primarily of lymphatics and lymph-glands, and not of the skin. Moderate to severe constitutional symptoms like fever, multiple arthralgia and rarely splenomegaly may be associated.

**Warty growths** *Verruca acuminata* (so-called condyloma acuminata) is a virus papilloma of the genital region giving rise to profuse cauliflower-like multiple growths (Fig 8 13). It is not a manifestation of syphilis but may occur especially in a female suffering from gonococcal infection, because of the excessive moisture and sodden condition of the parts. Condyloma acuminata may be mistaken for condylomata lata (Fig 8 14) which are hyperplastic papular growths seen round the anus and genitals. They are usually flat-topped with flat indurated bases.

**Non-venereal ulcers** **BALANITIS** Inflammatory redness of the corona, with or without ulceration and inguinal lymphadenopathy, may result from phimosis, lack of hygiene or local infection. It may be secondary to syphilitic chancre, chancroid or gonorrhoea.

**EPIDERMOID CARCINOMA**, a commonly occurring malignant tumour of the glans penis, is usually of the nature of a chronic hyperplastic indurated ulcer with everted edges and associated with hard lymphnodes.

**Priapism** A spontaneous and painful erection of the penis, over which the patient has no control, may arise in case of spinal cord lesions in association with paraplegia-in-flexion, whether due to spinal cord injury, tumour or myelitis, chronic myeloid leukaemia or local lesions of the corpora cavernosa penis. It may also be caused by lesions affecting the brain stem or tumor of the posterior fossa.

**Tumours** Benign tumours, such as sebaceous cysts and papillomata are fairly frequently observed over the skin of the penis and should cause no alarm.

**Hypospadias**. A congenital deficiency of the posterior wall of the urethra resulting in a chronically malformed short and downwardly curved penis (Fig 8 15).

**Underdeveloped genitalia** Smallness of the genitals is seen in hypogonadotropic eunuchoid (Fig 8 16), and in Frohlich's syndrome.

## SCROTUM AND TESTES

The contents of the scrotum and particularly the two testicles and epididymis are carefully palpated, between the thumb and fingers, for size, shape, consist-



ency, tenderness, weight and tumours. A comparative palpation of the two sides, being of value, should always be carried out. Palpation may prove difficult or impossible in case of massive oedema of the scrotal wall or hydrocele.

**Oedema** Oedema of the scrotum and penis, at times massive with the penis submerged within the contents of the scrotum, may result from generalized anasarca, as in congestive cardiac failure, wet beri-beri or renal oedema, venous or lymphatic obstruction, as in inferior vena caval thrombosis, or filariasis (Fig 8 17), or some acute inflammatory process, such as erysipelas of the scrotal skin.

**Hydrocele** An accumulation of serous fluid within the tunica vaginalis may be revealed by the presence of a tense, smooth, pyriform, slightly fluctuant, non-tender, non-inflammatory, swelling of the scrotum. Usually flat or dull to percussion, the characteristic translucence of a hydrocele can be demonstrated by transillumination with the aid of a pocket torch. Usually chronic and of obscure aetiology, a hydrocele may arise acutely after an injury or in association with an acute orchitis or epididymitis.

**Scrotal hernia** Liable to be mistaken for a hydrocele, a hernia that has descended into the scrotum can be distinguished by its being tympanitic to percussion, its reducibility and impulse on coughing.

**Varicocele** A mass of tortuous and dilated veins usually on the left side resulting from varicosity of the spermatic plexus, frequently associated with a dragging sensation and giving a bag of worms feeling on palpation. Occasionally, a varicocele is indicative of renal malignancy.

**Spermatocele** A small or large cyst, or collection of cysts arising from the upper pole of the epididymis and containing milky fluid with spermatozoa, can be distinguished from a hydrocele by lack of translucence, exact site of origin and paracentesis with examination of the fluid.

**Epididymitis** Acute epididymitis, a fairly common complication of gonorrhoea, is associated with painful, tender enlargement of the epididymis, swelling and redness of the scrotal wall and, at times, acute hydrocele and inflammation of the spermatic cord. Chronic epididymitis, usually secondary to tuberculosis or prostatic inflammation, is characterized by a slowly progressive, nodular, painless enlargement of the epididymis on one or both sides.

**Orchitis** Acute orchitis, with rapid, painful and tender swelling of one or both testes, may be noted in case of acute gonorrhoea, mumps, infectious fever or injury and usually results in testicular atrophy with impotence. Chronic orchitis, usually unilateral and associated with painless, non-tender enlargement of one or other testicle with loss of normal sensitivity, may arise in case of syphilis, tuberculosis or gonorrhoea.

**Undescended testicle** (Cryptorchism) A congenitally defective descent of one or both testicles may result in cryptorchism, with the undescended testicle either in the inguinal canal or groin and palpable, or intra-abdominal and

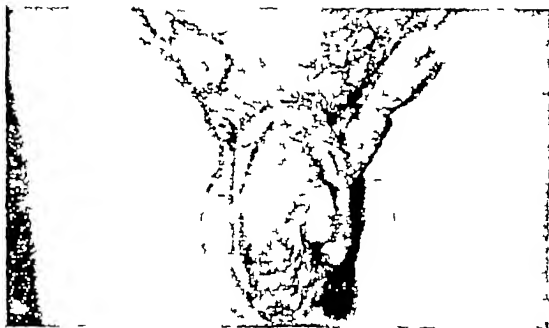


Fig 8 11 Granuloma venereum

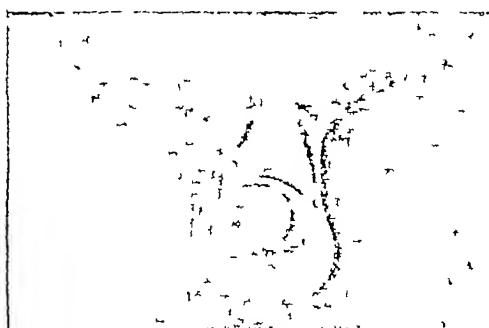


Fig 8 12 Lymphogranuloma inguinale



Fig 8 13 Verruca (condyloma)  
accuminata



Fig 8 14 Condyloma lata (syphilitic  
condyloma)



Fig 8 15 Hypospadias with bifid scrotum

Hypogonadism  
 il genitalia en  
 casts and scanty  
 ublic hair

Fig. 817 Elephantiasis of scrotum and skin of penis

Fig. 818 Massive elephantiasis of the vulvae

Fig. 819A Enlarged clitoris in adrenogenital syndrome

Fig 819B Note the hirsutism of the face

impalpable. The condition may be complicated by testicular atrophy, formation of hernia or malignant degeneration (when intra-abdominal).

**Testicular atrophy.** Small and shrunken testicles and epididymis, with atrophy, may either result from infectious fevers, such as mumps, injuries, torsion of the spermatic cord, or represent a congenital malformation.

**Testicular tumours.** Usually of the nature of adenocarcinoma, seminoma, teratoma or chorionepithelioma, malignant tumours of the testicle are hard, painless and non-tender swellings with late involvement of skin and inguinal lymphnodes but early metastases within the pelvic lymphnodes, liver and lungs. Painful swelling of the breasts (gynaecomastia) may accompany such tumours.

### Prostate gland

**PROSTATITIS.** *Acute prostatitis* or sudden swelling of the prostate gland with intense pain in the genital area, high fever, disturbances of micturition and painful defaecation, and with tendency to abscess formation, may result from acute gonorrhoea, cystitis, pyelonephritis or infection elsewhere in the body. *Chronic prostatitis*, which is secondary to acute inflammation of the prostate gland, may be entirely symptom-free and is usually characterized by a soft or hard, non-fluctuating moderately enlarged prostate on rectal examination.

**BENIGN HYPERTROPHY.** Common in elderly men and associated with urinary disturbances, such as difficult micturition, frequency, nocturia, dribbling and haematuria, benign prostatic hypertrophy usually reveals itself, on rectal examination, as a smooth, soft, enlarged, non-adherent prostate gland. When enlargement is confined to the median lobe of the prostate gland, the condition is likely to be missed on palpation, although capable of leading to acute retention of urine.

**CARCINOMA OF PROSTATE.** The commonest malignancy of the genito-urinary tract and at times difficult to distinguish from benign hypertrophy, carcinoma of the prostate gland, which is either symptom free or associated with pains in the legs or sacral area or haematuria, usually manifests itself on rectal examination, as a remarkably firm or hard, nodular and fixed enlargement of the prostate gland. Signs of diagnostic value are the stony hardness of the prostate gland, its adherence to adjacent tissues, early development of bone metastasis and high levels of alkaline phosphatase in the blood.

### FEMALE GENITALIA

Examination of female genital organs, particularly of the vulva, vagina, cervix and uterus, should be carried out routinely, except in the case of virgins. Vaginal examination should be regarded as an integral part of examination of a female patient. Involvement of female genital organs, although at times asymptomatic, may be suggested by the presence of certain symptoms, such as local pain, dysmenorrhoea or amenorrhoea, leucorrhoea or mucopurulent discharge and pruritus vulvae.

**METHOD OF INSPECTION.** This may be carried out either in the lithotomy position, with the patient supine, thighs and legs flexed and knees wide apart,

or with the patient lying on her side, lower leg extended and upper one fixed at the hip and knee. The following are then systematically inspected: vulva, labia, clitoris, urethral aperture, vaginal orifice, hymen and perineum. With the aid of a vaginal speculum and proper illumination, it is possible to observe the vaginal walls and cervix, a look-out being kept for abnormalities of size, colour, consistency and evidences of discharge, bleeding, tumours and ulcerations.

**VAGINAL EXAMINATION (Bimanual method of palpation)** With the patient in the lithotomy position and urinary bladder empty, the examiner, standing on the patient's left, inserts the index finger of his left hand slowly and with care into the vagina, whilst palpating the lower abdomen from above, with his right hand placed above the symphysis pubis. By a proper coordination of the two hands, it is then possible to palpate bimanually and systematically vaginal walls, cervix uteri, uterus, ovaries and tubes, for evidence of disease or abnormality. Vaginal examination should always be followed by rectal examination (to confirm the findings obtained) and by careful palpation of the inguinal lymphnodes.

**The vulva** Various lesions, such as venereal and non-venereal ulcers, condylomata, pediculosis, cysts and tumours, described in the case of males, may also be observed in the case of female patients.

**LEUKOPLAKIA** Usually secondary to long-standing pruritus and particularly common after the menopause, leukoplakic vulvitis is characterized by the appearance on the vulva or perineum of multiple dirty white, grey or bluish-patches, which may subsequently develop into kraurosis or cancer of the vulva.

**KRAUROSIS** A shrinking or atrophy of the labia and clitoris, with a cracked and leathery parchment-like thinning of skin, may be encountered after the menopause particularly in women with longstanding leukoplakia and pruritus.

**OEDEMA OF VULVA** Usually part of a generalized oedema or anasarca, vulval oedema may result from local infection, carcinoma of the uterus, intra-abdominal tumour or pregnancy. Obstruction of lymphatic return leads to elephantiasis of the vulva (Fig. 8 18).

**ENLARGED CLITORIS** A hypertrophy of the clitoris is usually of endocrine origin and may be noted in case of Cushing's syndrome, adrenogenital syndrome (Fig. 8 19A) or, rarely ovarian tumour.

**The vagina** **VAGINITIS** Inflammation of the vagina (acute, subacute or chronic) may result from a variety of causes, such as gonorrhoea, trichomonas infection, moniliasis, and usually manifests itself as a blotchy red swelling of the vaginal mucosa with purulent or watery discharge.

**CYSTOCELE OR RECTOCELE.** A downward and forward bulging of the anterior or posterior wall of the vagina in connection with the urinary bladder in front or the rectum behind.

**The cervix and uterus** **CARCINOMA OF CERVIX** The commonest of all tumours affecting the female genitalia, cervical carcinoma may be suggested by the appearance of abnormal vaginal bleeding or discharge. Frequently associated with a hard and nodular bleeding cervix uteri, the condition may appear as a patch of leukoplakia or a red hard and nodular mass.

**CARCINOMA OF BODY OF UTERUS** Usually developing after the menopause, the condition may first manifest itself through a watery discharge or vaginal bleeding. Vaginal examination may reveal a symmetrically enlarged or normal sized uterus, with or without metastatic deposits, in the ovaries, cervix or vagina.

**UTERINE FIBROIDS** Excessive and irregular bleeding from the vagina, with or without a foul and watery discharge, when associated with an enlarged, hard or nodular uterus and anaemia, should suggest the possibility of fibroid tumour or tumours.

**ENLARGED UTERUS** An enlargement of the uterus may result from a variety of causes, physiological and pathological, including pregnancy, fibroid tumour, endometrioma, carcinoma and subinvolution.

**ABNORMAL POSITION OF UTERUS** The uterus may display anteflexion or forward angulation, retroversion or backward angulation, prolapse or downward displacement and procidentia or massive prolapse.

**The Fallopian tubes and uterus** **ACUTE SALPINGITIS** Frequently gonococcal in origin and at times without a history of sexual exposure or venereal infection, acute salpingitis is characterized by bilateral or unilateral pain in the lower abdomen, with tenderness, fever, nausea, vomiting and backache. Vaginal examination discloses as a rule a red and swollen cervix with a muco-purulent discharge, extreme tenderness to touch of the uterine adnexa and the presence of a small or large tender mass on one or both sides. Although often related to the menses, of more acute and febrile onset and with pain and tenderness not confined to the right side of the abdomen, acute salpingitis may be very difficult to distinguish from acute appendicitis.

**CHRONIC SALPINGO-OOPHORITIS** Chronic pelvic infection, usually secondary to gonococcal or puerperal infection, may be either symptom-free or exhibit low abdominal pain, pelvic discomfort and menorrhagia. Vaginal examination may disclose a cervicitis with palpable thickening or nodularity of the adnexa. Careful palpation may disclose a hydrosalpinx, pyosalpinx or pelvic abscess. In the presence of pulmonary tuberculosis, the possibility of tuberculous salpingitis should be considered.

**PELVIC PERITONITIS** Acute or chronic peritonitis, localized to the pelvic regions, may follow an attack of acute salpingitis, puerperal infection or pelvic appendicitis. It is accompanied by pain and tenderness in the regions of the lower abdomen, vagina and rectum.

**TUBAL GESTATION (Tubal pregnancy)** This may be suggested by sudden vaginal bleeding and unilateral lower abdominal pain arising in a woman in conjunction with an amenorrhoea of one or two months duration. Severe unilateral lower abdominal pain, signs of massive internal haemorrhage and vaginal bleeding arising suddenly in such a case, in association with a tender, palpable mass on one or other side on vaginal examination and a positive Cullen's sign (blueness of the umbilicus) should immediately suggest the possibility of the dreaded complication of tubal rupture or abortion.

**OVARIAN TUMOUR** This may be of any size, shape or consistency, benign or malignant, and symptom-free or associated with menstrual disturbances, low back-ache, low-grade pyrexia and bearing down pain. When small, an ovarian tumour is easy to miss. A large ovarian tumour, on the other hand, may be mistaken for ascites, fibroid tumour or pregnancy.

# 9

## The Cardiovascular System

**Anatomy** The cardiovascular or circulatory system, which is concerned with the circulation of blood, is composed of two main elements, viz., the heart or central pump and the blood vessels or distributing channels

The heart is a hollow muscular organ, suspended in the middle mediastinum, within a pericardial sac and between the two lungs. Normally, two-thirds of the heart lies to the left and a third to the right of the midline, its long axis is directed obliquely downwards, forwards and to the left. The base is mainly formed by the left atrium and the apex exclusively by the left ventricle.

The distributing system of blood vessels, starting from the ventricle, is systematically composed of arteries (large and small), arterioles, capillaries, venules and veins (small and large) leading the blood back into the heart, through the corresponding atrium.

First described by Erasistratus in Greece about 250 B.C. and accurately portrayed by Leonardo da Vinci in his many drawings, the *valves* of the heart serve to maintain a unidirectional (or one-way) flow of blood in the circulatory system. There are two atrioventricular (mitral and tricuspid) valves and two semilunar (pulmonary and aortic) valves in each heart. Since the production of heart sounds and murmurs is mainly dependant on the valves of the heart, their behaviour during health and disease merits careful study.

The pericardium, with its visceral and parietal layers, serves 3 main functions: it (1) encases the heart and pericardial fluid, (2) prevents excessive movement of the heart, and (3) prevents overdistension or excessive dilatation of cardiac chambers.

**Topographical anatomy** The left ventricle normally occupies but a narrow strip along the left border, whilst the right ventricle accounts for the major part of the anterior surface of the heart. A smaller area to its right is accounted for by the right atrium. The left atrium appendage, behind the second left intercostal space, is the highest part of the heart in the thorax, whilst the major part of the left atrium is situated behind the heart. The lower border of the third left costal cartilage overlies the pulmonary valve.

The precordial area or precordium is that portion of the anterior or front wall of the chest which overlies the heart.

**Important landmarks** Certain landmarks, both natural and artificial, are of value in the study of the cardiovascular system. The ribs and intercostal spaces (or interspaces), on either side, are useful *horizontal* landmarks. The ribs are usually counted downwards from the level of the second rib, which is in line with the angle of Louis or sternal angle (the prominent ridge at the junction of the manubrium sterni with the body of the sternum) and is therefore easily identified. Posteriorly, the ribs may be counted upwards from the twelfth rib (which is usually palpable), or from the seventh rib which lies at the level of the inferior angle of the scapula (with the arms hanging down by the sides).

As *vertical* landmarks a series of vertical lines are drawn or imagined on the chest. These are *midsternal* line drawn through the centre of the sternum, the *lateral sternal* or sternal line along the border of the sternum (on either side), the *midclavicular* line drawn midway between the centre of the suprasternal notch and the tip of the acromion process, the *parasternal* line midway between the lateral sternal and midclavicular lines, the *anterior axillary*, *mid-axillary* and *posterior axillary* lines drawn through the anterior fold, centre of the apex of the axilla and posterior fold of the axilla respectively, the *scapular* line through the inferior angle of the scapula, and the *midspinal* line along the centre of the spinal column. The *mammary* (or mammillary) line, drawn through the nipple, being undependable, has now been superseded by the mid-clavicular line.

## THE ARTERIAL PULSE

**Importance** Palpation of the radial pulse is an important and essential part of examination. It affords (1) valuable information about the condition of the arterial walls, (2) a rough estimate of the systolic and diastolic blood pressures, (3) information regarding the state of the heart and circulation, (4) help in the detection and diagnosis of arrhythmias of the heart, and (5) diagnostic information about entities, such as aortic regurgitation (water-hammer pulse) and left ventricular failure (pulsus alternans). It has been called the "mirror" or "index" of the heart.

**Mechanism** The pulse does not represent the movement of the blood column ejected from the heart by systolic contraction, but a *wave* (pulse wave), which, after being produced by cardiac systole, traverses or advances through the arterial tree in a peripheral direction, at a rate much higher than that of the column of blood. It arrives at the wrist long before the column of blood ejected by the heart.

**Method** It is essential as a routine to feel not only the radial pulses but also the other peripheral pulses. The pulse is usually felt at the wrist and over the radial artery, because of its superficial position and ease of palpability.

**Radial pulse** The radial artery is situated slightly medial to the styloid process of the radius, on the anterior aspect of the wrist, and is best felt with the subject's forearm slightly pronated and wrist somewhat flexed. Both



the radial arteries should be palpated simultaneously to *detect inequalities* of pulse on the two sides (suggestive of certain diseases) Difficulty in locating the radial artery may be due to (1) an aberrant or anomalous course of the artery, or (2) an unusually small radial artery

*Brachial pulse* The *brachial* artery, with the elbow flexed, should be palpated either with the thumb or fingers of the right hand Thickening of the arterial wall, visible pulsations (or "dance") of the arteries and pulse characteristics are often better appreciated at the elbow than wrist

*Carotid pulse* The carotid artery since it is the nearest palpable artery to the arch of the aorta it gives the best indication of the pulse wave form The artery is palpated with the thumb (left thumb for palpating the right carotid) or the finger tips placed at the upper end of the thyroid cartilage along the medial border of the sternomastoid muscle

The following is the importance of palpating the carotid pulse (1) The carotid pulse is a better guide to the timing of auscultatory events in the cardiac cycle than the radial (2) Absent or unequal pulsations on the two sides usually results from atherosclerosis and is indicative of carotid artery stenosis (3) In severe aortic stenosis the pulse is often small and prolonged (4) Pulsus alternans can be best felt in the carotid pulse (5) Carotid compression may be used to slow the heart rate and thus facilitate timing in auscultation, to separate the two components of gallop rhythm, to terminate an attack of paroxysmal tachycardia or to temporarily change the degree of block in atrial flutter, or to confirm or exclude that hypersensitivity of carotid sinus may be the cause of syncopal attacks (6) Carotid sinus compression may terminate an attack of angina pectoris

*Femoral pulse* The femoral artery should be felt with the leg slightly abducted and the foot externally rotated and the examining hand pressed firmly into the groin Inequality or diminution of the pulse should be noted and the time of appearance should be compared with that of the radial pulse by palpating both simultaneously Weak, delayed or absent pulsation of the femoral artery is suggestive of coarctation of aorta, occlusive disease of bifurcation of aorta, common iliac or external iliac artery, or middle aortic syndrome

*Popliteal pulse* The popliteal artery can be palpated by putting the patient in the supine position and both the hands of the examiner encircling and supporting the knee from each side

*Posterior tibial pulse* The artery can be felt in the groove between the medial malleolus and tendo Achilles, the fingers of the examiner curving around the ankle

*Dorsalis pedis pulse* The artery can be palpated just lateral to the extensor hallucis tendon the examiner placing his finger tips across the dorsum of the foot

**Characteristics of the pulse.** A systematic observation should be made of the following characteristics of the radial pulse

1 **RATE** The pulse rate should be counted for one full minute. Normal pulse rate varies between 60 to 80 per minute. Increase in pulse rate is labelled tachycardia and decrease bradycardia.

*Pulse deficit* The pulse rate corresponds to the heart rate, except in case of extrasystoles or atrial fibrillation, where a marked difference, so called pulse deficit, may be observed. In such cases, some of the heart beats are not registered at the wrist at all, the pulse deficit being at times as high as fifty or more per minute.

2 **RHYTHM** The pulse at the wrist is usually regular in rhythm and force. An irregular pulse, with an unequal spacing of the beats, may be due to (i) sinus arrhythmia, (ii) extrasystoles (premature or ectopic beats), (iii) atrial fibrillation, (iv) paroxysmal atrial tachycardia with block, (v) atrial flutter with varying block, (vi) partial heart block with irregularly dropped beats.

In *sinus arrhythmia* (See p 202) there is a phasic irregularity dependent on the respiratory phases, there being a gradual waxing or acceleration during inspiration and a waning or slowing during expiration. Being common in childhood, because of overaction of the vagus nerve, it is sometimes referred to as "juvenile irregularity of the heart".

An intermittent dropping out of beats at the pulse is referred to as an "intermittent pulse". The beats may be dropped regularly (as in *pulsus bigeminus* or coupling, every two beats being followed by a pause, or *pulsus trigeminus*, with a drop after every three beats), or dropped irregularly. When a pulse is totally irregular or irregularly irregular (*pulsus irregularis perpetuus* or *delirium cordis*), there being no suggestion of an underlying dominant rhythm, the condition is referred to as atrial fibrillation. A pulse deficit is a constant feature of the condition. An intermittent pulse may be due to partial heart block (with dropped heart beats). Whilst the former irregularity tends to disappear on exercise or artificially induced tachycardia, acceleration of the heart rate increases the latter defect.

3 **VOLUME** The degree of expansion (or amplitude), displayed by an artery between its diastolic or empty state and systolic or filled state, is called volume, size, amplitude or expansion. Proportional to the pulse pressure, it depends on the state of contraction or relaxation of the arteries, and the force of contraction of the heart.

*Hypokinetic pulse* (weak pulse) A small pulse signifies a narrow pulse pressure and is a result of a low cardiac output as in shock, congestive cardiac failure, hypovolaemia, cardiac tamponade, chronic constrictive pericarditis, myocarditis or cardiomyopathy, valvular disease such as aortic outflow tract obstruction, mitral stenosis or aortic disease like coarctation or aortic arch syndrome.

**Hyperkinetic pulse** (bounding pulse) It usually implies a high pulse pressure and is the result of varying contribution of increased stroke volume, increased cardiac output and lowered peripheral resistance, e.g. in anxiety, exercise, complete heart block or aortic regurgitation, decreased peripheral resistance e.g. fever, anaemia, thyrotoxicosis, hyperkinetic heart syndrome, A-V fistula, Paget's disease, beriberi, cirrhosis of liver or decreased distensibility of the arterial system as in atherosclerosis, hypertension or coarctation of aorta

**4 FORCE** The compressibility of a pulse or its force affords a rough measure of the systolic blood pressure, and may be assessed in one of two ways (i) The three middle fingers of the examiner's right hand being placed on the radial artery, the distal finger (i.e. nearest to the patient's wrist) is kept firmly pressed (in order to prevent pulsation from the ulnar artery being conducted into the radial artery through the palmar arch) The middle finger (palpating finger) is placed with sufficient force over the artery to get the maximum pulsation of the radial artery The proximal finger (compressing finger) then gradually compresses the artery above this point, until the pulse can no longer be felt by the middle finger The pressure required (with the proximal finger) to obliterate the pulse (under the middle finger) is proportional to the systolic blood pressure (ii) A less cumbersome method is to gradually compress the right brachial artery with the right thumb, whilst palpating the radial artery on the same side with the fingers of the left hand The degree of compression of the brachial artery required to obliterate the radial pulse gives a fair measure of the systolic pressure

**5 TENSION** This corresponds to the diastolic blood pressure and may be gauged in one of two ways (i) The state of the artery between pulse beats is noted for its degree of hardness or softness, a rough measure of its tension In a low tension pulse (*pulsus mollis*), the vessel is soft or unpalpable between beats, whilst in a high tension pulse (*pulsus durus*), it stands out rigidly like a cord or tendon under the fingers (ii) Another simple method of measuring tension is to palpate the pulse with light, medium and then heavy pressure to determine which of the three yields the best pulsation In low tension, the pulsation is best felt with light pressure, in high tension with heavy digital pressure, a normal pulse is best felt with medium pressure.

**6 FORM OR CONTOUR OF THE PULSE WAVE** This is a palpatory estimation of the arteriogram Although better appreciated with a sphygmograph, the form or contour of the wave can often be assessed with a trained finger The pulse wave is studied from the points of view of (i) rise or upstroke, (ii) summit, plateau or peak, and (iii) fall or downstroke The rise of the pulse may be rapid or slow, the summit well-sustained (plateau-like) or ill-sustained (or peaked), and the fall sudden or gradual A pulse with a quick rise and a quick fall is called *pulsus celer*, and is typical of aortic regurgitation A slow rise and a slow fall (*pulsus tardus*) are typical of aortic stenosis

Normally, the dicrotic wave on the descending limb of the pulse wave is not palpable. When it becomes clearly palpable, as in states of low tension and during typhoid and other fevers, the pulse imparts a double impact with

each beat (*pulsus dicroticus* or *dicrotic pulse*) It is indicative of relaxed arteries, low peripheral resistance and low blood pressure

7 **EQUALITY** Weak or absent pulsation of one or both *radial* arteries may be indicative of (i) Anomalous or aberrant course of a radial artery is the commonest cause, (the brachial artery pulsations and blood pressure readings on the affected side are normal in such cases) (ii) Thrombosis or embolism of the brachial, axillary or subclavian artery (iii) Aortic arch syndrome or pulseless disease (iv) Aneurysm of aorta (v) Dissecting aneurysm of the aorta (vi) Coarctation of aorta (vii) Cervical rib (viii) Scalenus anticus syndrome (ix) Previous arterial catheterization (x) Supra-valvar aortic stenosis

8 **CONDITION OF ARTERIAL WALL** This can be estimated by flattening the artery by digital pressure the empty vessel then being palpated with the fingers sliding up and down and sideways Whilst a normal artery is not palpable, an atherosclerotic vessel may be either palpable but smooth and muscular, or hard, rigid and tube like (pipe-stem, metal wire or whip cord) or goose neck or tortuous

9 **RADIO-FEMORAL DELAY** In coarctation of the aorta the femoral pulse as compared to the radial (or brachial pulse) may be appreciably delayed

#### Abnormal pulses·

**Bounding pulse** (*Collapsing pulse* or *pulsus celer*) There is a sharp up-stroke, a high peak and a sudden downstroke, features accentuated by raising the arm high It is found in hyperkinetic circulatory states (e g , fever, thyrotoxicosis, emotional excitement) and in states of peripheral vasodilatation It is indicative of a low peripheral resistance, high pulse pressure and increased blood flow It gives the erroneous impression of being a "strong" pulse

**Corrigan or water-hammer pulse** (See p 202) This is an exaggerated form of bounding or collapsing pulse It is a combination of *pulsus celer* and *pulsus magnus* (i e , a quick rise and fall, high pulse pressure) Described by Corrigan and Vieussens, and variously described as the "water-hammer", collapsing or jerking pulse, this variety of pulse is almost diagnostic of aortic regurgitation

The term "water-hammer" comes from a nineteenth century toy, the "water-hammer", This was a long, hermetically sealed glass tube containing water and vacuum On inversion the water would suddenly drop through the vacuum, imparting a palpable shock or knock

A water-hammer pulse indicates a low filling resistance in the blood vessels into which the left ventricle pumps out blood This may be due to (1) Physiological causes such as exercise, heat, emotion, alcohol, pregnancy (2) Hyperkinetic circulatory states e g , anaemia, thyrotoxicosis, Paget's disease of bone, hepatic disease, beriberi, and some cases of anoxic cor pulmonale (3) A leak in the arterial side of the circulation, e g aortic incompetence, patent ductus arteriosus, arteriovenous fistula, large ventricular septal defect, and

to a lesser degree in mitral incompetence (4) In complete heart block a water-hammer pulse may occur because of the fact that with each systole a comparatively large volume of blood is pumped into a relatively empty reservoir

The typical character of the pulse is displayed to advantage by grasping the forearm of the patient with the entire hand (the flexor aspect being covered with the palm) and raising it about the level of the patient's head

The characteristic sudden, forceful and jerky rise of the Corrigan pulse is due to the rapid filling of the radial artery caused by an extra large amount of blood pushed by the distended left ventricle during systole into relatively empty arterial vessels The collapsing character or the sudden downstroke of the pulse may be due partly to the sudden fall of pressure in the aorta due to regurgitation of blood into the left ventricle through a leaky valve during diastole, and partly to the rapid emptying of the arterial system because of the markedly increased velocity of the blood stream The accentuation of the water-hammer character of the pulse, by palpating the wrist with the patient's arm elevated, is due partly to the effect of gravity emptying the peripheral artery, thus accentuating the pulse pressure, and partly to the fact that in this position, the radial artery is more in line with the outflow stream from the aorta

**Dicrotic pulse** There is a second small wave (dicrotic or diastolic wave) following the peak of the main wave A dicrotic pulse may be found in debilitating fevers, dehydration, advanced myocardial failure It may be observed in early postoperative days following aortic valve replacement for AR

**Pulsus parvus** (parvus=small) The term suggests that the amplitude of the pulse is small and it is commonly encountered in presence of a low cardiac output A pulse of small amplitude usually associated with tachycardia is labelled as a thready pulse

Pulsus parvus may be (1) Physiological due to vasoconstriction from cold or anxiety (2) Partial occlusion of a vessel (3) Coarctation of aorta (4) Severe hypotension (5) Aortic stenosis (6) Myocardial infarction (7) Severe pulmonary hypertension (8) Pulmonary stenosis of severe degree

**Pulsus parvus at tardus** This is a small (parvus), slow rising and slow falling (tardus) pulse and is characteristic of severe aortic stenosis

**Plateau pulse** If the delayed systolic peak in aortic stenosis is sustained it is labelled a plateau pulse

**Anacrotic pulse** (ana=up, crotos=beat) When a small wave is felt along the upstroke (analogous to the dicrotic wave of the dicrotic pulse) the pulse is called anacrotic The anacrotic peak is due to the percussion wave (early systolic wave) of ventricular systole transmitted through the stenosed aortic valve in aortic stenosis

**Pulsus bisferiens** (bis=twice, feriens=beat) It is a double pulse with two peaks occurring during systole It is found with combined moderate aortic stenosis and severe aortic regurgitation Moderate AS causes an extra high velocity jet to be shot out and this is exaggerated by an increased flow due to severe AR during previous diastole At the peak rate of flow there

is a Bernoulli effect (lateral pressure is inversely proportional to the velocity of flow through a tube) on the walls of the ascending aorta, causing a sudden fall in pressure on the inner side of the aortic wall. Pulsus bisferiens may also be detected in hypertrophic obstructive cardiomyopathy (HOCM). Here there is initially no obstruction to outflow, the obstruction appears later in systole as the mitral valve begins to approximate the hypertrophied septal area. There is a sharp drop in pressure followed by a secondary rise to overcome the obstruction.

**Jerky pulse** This refers to a combination of a small volume pulse and a collapsing pulse. There is a rapid upstroke and a quick fall off early in systole. It occurs when the left ventricle empties rapidly in systole and may be encountered in severe M R, V S D and HOCM.

**Pulsus alternans** There is a regular alternation in the size of the beats, the beats being regularly spaced but alternately large and small. It is encountered in (1) left ventricular failure where it is considered an ominous sign, (2) cardiac arrhythmias (e.g. paroxysmal tachycardia or after extrasystole), in which case it is not significant. It is best detected over the carotid artery. It can rarely be appreciated at the wrist but may be brought out by compressing the brachial artery above. Rarely the weak pulse may not be palpable at the wrist (total alternans).

The condition is not associated with electrical alternans (in the electrocardiogram), the blood pressure readings of alternate beats in pulsus alternans may differ by 10 to 30 mm. Whilst in pulsus bigeminus the weak beat is close to the preceding normal beat, in pulsus alternans the beats are either quite evenly spaced or rarely the weak beat is somewhat closer to the succeeding than the preceding beat.

Pulsus alternans is usually attributed to severe damage, disease or fatigue of the ventricular muscle, resulting in uneven excitability or refractoriness of the muscle fibres. The alternating strength of ventricular contraction is due to the fact that whilst during the strong beat there are fewer refractory fibres which fail to contract, during the weak beat a larger number of fibres are refractory. Since the pulse reflects ventricular strength of contraction, it becomes alternately strong and weak.

**Pulsus paradoxus** In normal individuals the systolic B P drops by 2-4 mm Hg with normal inspiration and upto 10 mm Hg during deep inspiration because (a) the lung capacity increases during inspiration and there is a greater negative intrathoracic pressure resulting in pooling of blood in pulmonary vessels and hence less blood goes to the left side of the heart, (b) Intrathoracic pressure decreases on inspiration and since the aorta is an intrathoracic structure its pressure also falls. When the systolic pressure falls more than 10 mm Hg during inspiration the pulse is referred to as pulsus paradoxus.

Actually the term pulsus paradoxus is a misnomer because normally the systolic pressure decreases with inspiration. The term was introduced by Kussmaul who found a marked drop of blood pressure on inspiration in patients with constrictive pericarditis, observed that the heart beat did not change in any manner despite drop of pulse with inspiration.

CAUSES (1) *Cardiac*—Constrictive pericarditis, cardiac tamponade, pericardial effusion The systolic B P as well as the pulse pressure decrease on inspiration because of marked drop in cardiac output. (2) *Pulmonary*—



NORMAL



PULSUS BIGEMINUS



PULSE IN A. FIBRILLATION



DICROTIC



ANACROTIC



WATERHAMMER



PULSUS TARDUS



BISFERIENS



PULSUS ALTERNANS



THREADY



SINUS ARRHYTHMIA



INSPIRATION

INSPIRATION

EXPIRATION



PULSUS PARADOXUS

Diagrammatic representation of various types of pulse tracings

(a) Bronchospasm as in severe asthma or emphysema because if expiration raises the intrathoracic pressure too high as a result of bronchospasm, then inspiration will by contrast seem to lower the systolic pressure excessively  
 (b) Obstruction of superior vena cava This causes limitation of inspiratory increase in blood flow to right atrium (3) *Miscellaneous*—(a) Obesity possibly due to excessive compression of inferior vena cava at the thoracic inlet (b) Hypovolaemic shock because of poor filling of lungs and right ventricle in inspiration because peripheral venous reservoirs withhold more blood than normal during inspiration

**UNILATERAL PULSUS PARADOXUS** may result from compression of one anomalous subclavian artery

**REVERSED PULSUS PARADOXUS** Here there is inspiratory rise in arterial systolic and diastolic pressures It may be encountered in (1) Hypertrophic obstructive cardiomyopathy because of decrease in left ventricular volume on expiration from a Valsalva-like effect, thus lowering the blood pressure as compared to systolic levels (2) During intermittent positive pressure breathing (IPBB) since blood is squeezed out of pulmonary capillaries and venules during positive pressure inspiration, thus augmenting left ventricular stroke volume and blood pressure (3) Isometric ventricular rhythm possibly because the increased heart rate during inspiration permits atrial 'capture' (for a few beats) increasing left ventricular volume and output during inspiration

**CAPILLARY PULSATION** In aortic regurgitation and conditions associated with peripheral vasodilatation, capillary pulsation can be elicited in various ways. (1) Gentle pressure is applied at the tip of the nail and the nail bed observed for a rhythmic flush It can also be seen by transilluminating the ear tip or finger or thumb tip by a torch light, shading the part observed with the flexed fingers (2) By gently compressing the mucous membrane of the lips by a glass slide (3) By rubbing the skin of the forehead and observing the alternate blushing and blanching of the hyperaemic area

The nail fold capillaries can be best seen through a monocular compound microscope The light source should be intense and directed towards the nail bed at an angle of from 45 to 60°

**DIGITAL THROBBING** This has the same significance as capillary pulse It can be detected if the examiner approximates the tips of the fingers of his right hand to those of the patient's right hand

## BLOOD PRESSURE

**Sphygmomanometry.** Sphygmomanometry or measurement of the arterial blood pressure with a special instrument called the sphygmomanometer has become an integral part in the routine examination of the cardiovascular system The term blood pressure, when so used, usually refers to the systemic arterial blood pressure in the brachial artery

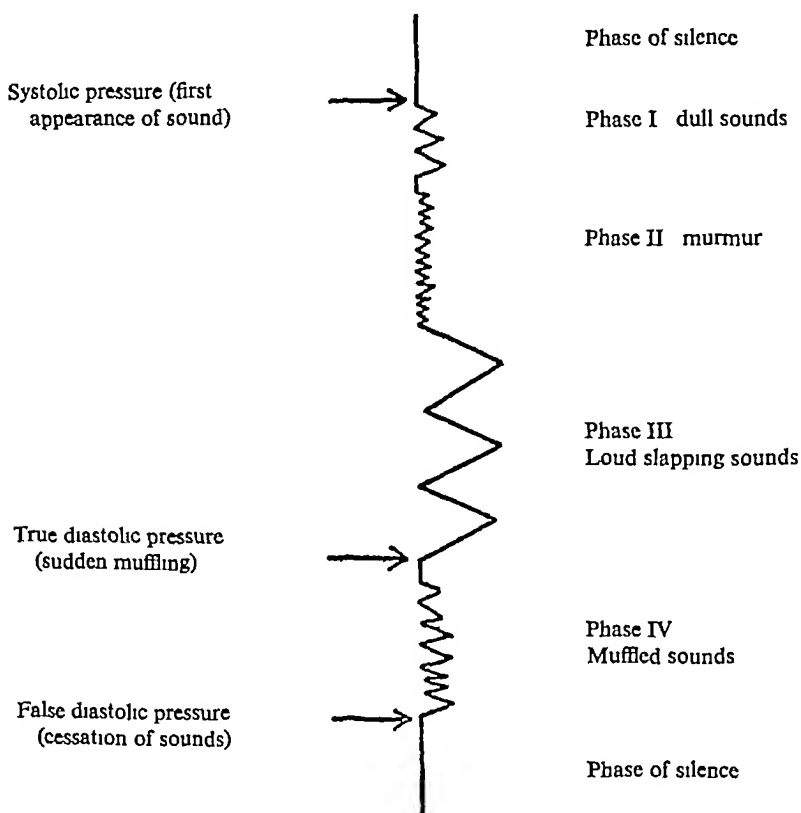
**HISTORICAL** The arterial blood pressure was first measured by the direct method, in a live mare by Rev Stephen Hales in 1733, a brass tube being inserted in the left crural artery



Von Basch (1810) devised the first practical instrument for measuring the blood pressure in humans. This was improved upon by Potain, Riva-Rocci and others. The auscultatory method, described by Korotkow in 1905, gained universal acceptance.

The clinical method of estimating pressures by a study of the pulse is unreliable and has been superseded by the instrumental or *sphygmomanometric* method of measuring the pressure. The oscillometric method is less convenient and more difficult of interpretation.

Diagram illustrating the auscultatory technique of measurement of arterial blood pressure



**Sphygmomanometric equipment** Two types of sphygmomanometers are in use the mercurial type and the aneroid type. The former is more accurate and lasting, requires only a few calibrations, but is somewhat cumbersome. Of the two, the mercurial type is recommended for routine use.

**Preparation** For sphygmomanometry, the patient should be comfortably seated or recumbent, completely relaxed and neither tired nor emotionally upset. The arm is freed of clothing up to the shoulder and supported comfortably, with the elbow flexed and at the level of the heart.

The cuff (of standard size) with a rubber bag at least 12 cm wide, after complete deflation, is snugly applied over the upper arm, with the middle of the rubber bag over the brachial artery, on the inner aspect of the arm, and the lower border of the cuff one inch above the bend of the elbow. The cuff must not show any bulging or displacement on inflation.

**Palpatory technique.** With the fingers of one hand on the patient's radial (or brachial) pulse, the examiner quickly inflates the cuff with the other hand until the arterial pulse is obliterated. The cuff is then gradually deflated by releasing the air through the exhaust valve, and the point at which the arterial pulse reappears is recorded at the systolic pressure. Too rapid or too slow a deflation of the cuff should be avoided. A very rough estimation of the diastolic pressure may be obtained by palpating the brachial artery during further deflation of the cuff after the determination of the systolic level. The pulse first assumes a water-hammer quality and then suddenly reverts to its normal character. The diastolic pressure corresponds to the point at which the brachial pulse changes from water-hammer type to normal.

Although the palpatory method has been abandoned by some in favour of the auscultatory, a routine adoption of both methods is strongly urged, partly to check the results of one with other, and partly to prevent erroneous readings caused by the so-called "auscultatory gap" in the case of the auscultatory method.

**Auscultatory technique.** The chestpiece of the stethoscope is placed firmly but not forcibly over the brachial artery, below the cuff, and without touching it. The cuff is inflated to a pressure about 30 mm. Hg above the level of systolic pressure detected by the palpatory method and then gradually deflated. The highest point at which the first clear sound, followed by successive sounds (Korotkow's sounds), makes an appearance corresponds to the systolic pressure. With further deflation, the dull sounds are replaced by a murmur (phase II), followed in turn by loud, sharp, tapping sounds (phase III). The point at which (on further deflation) these loud sounds suddenly become muffled or "murmurish" (phase IV) is regarded by some as the true diastolic pressure. With further deflation, at a point usually 5 to 10 mm lower, the arterial sounds disappear completely. There is considerable difference of opinion regarding the correct end point for the diastolic pressure—whether it is the point of change from slapping to muffled sounds or the more simply determined point of cessation of all sounds that is the true diastolic pressure. The latter is simpler and sufficiently accurate for routine purposes, and is therefore recommended.

It has been suggested that in case of an appreciable difference between the two diastolic readings, both end points should be recorded thus 150/90-80 or 160/50-20.

When "total disappearance" of auscultatory sounds is used as a criterion for the diastolic blood pressure, the latter may be underestimated or even reported as "zero" in cases of aortic regurgitation, arteriovenous shunt or

hyperdynamic circulatory states, conditions associated with low diastolic and high pulse pressures. Direct arterial puncture may disclose the true diastolic pressure as being in the vicinity of 40 mm in such cases. When "muffling of sounds" is used as a guide to the diastolic level, inaccurate readings (with over-estimation of diastolic pressures) are likely to be reported in cases of high blood pressure and in some normal individuals.

*False or erroneous* blood pressure readings are usually indicative of defective technique. They may be caused by (1) Wrong width or size of the blood pressure cuff. Unduly small cuffs are likely to give higher than normal readings and *vice versa*. The recommended sizes for arm "cuffs" are 13 cm width for adults (standard size) and 3 to 9 cm for infants and children. "Cuffs" for the estimation of crural blood pressures (in the thigh) must be 18 to 20 cm wide. (2) Too loose or tight an application of the cuff. (3) Wrong placement of the inflatable part of the cuff (beyond the brachial artery). (4) Too high or too low a position of the arm, undue elevation being responsible for unduly low pressure readings. (5) The arm being held in the air and not supported or resting on bed or table. (6) Excessive pressure of the chestpiece on the brachial artery. (7) Excessive adiposity or thickness of the arm. (8) Excessively hardened or incompressible brachial artery. (9) Unduly low levels of diastolic pressure (as in aortic regurgitation) or of systolic and diastolic pressures (as in shock). (10) Determination of blood pressure in one arm only. Over 10 per cent of normal adults show blood pressure differences of from 20 to 30 mm Hg between the two arms, minor differences being far more common.

For recording the *femoral* or crural blood pressure, the technique is similar, but a wider and longer cuff is used, the patient adopts a prone position and the chestpiece is placed over the popliteal artery, in the popliteal space, 1 to 2 inches below the lower edge of the cuff. The femoral pressure is normally from 0 to 10 mm higher than the brachial.

In hypertension or aortic stenosis, a silent gap or auscultatory gap may be noted. During deflation of the cuff, the initial phase of sounds is followed by complete or relative silence (auscultatory gap) finally leading into loud tapping sounds. If the cuff is not inflated in such cases, to a level higher than the systolic pressure (as checked by the palpatory method), the systolic reading may be grossly underestimated and recorded at the point at which the auscultatory gap ends and the loud sounds of phase III begin.

In atrial fibrillation, the systolic pressure corresponds to the level at which the majority of sounds come through, whilst the diastolic corresponds to the level at which most beats become suddenly muffled. In atrial fibrillation, the readings are only approximate.

In the case of premature beats, the pressure readings should not be based on premature beats which give unduly low readings or on post-extrasystolic beats which give unduly high readings, but on normal beats.

In cases of left ventricular failure, *pulsus alternans* should be looked for sphygmomanometrically, as it may be detected earlier than at the wrist. With gradual deflation of the cuff,

the sounds, after appearing at the systolic level, suddenly show a doubling of rate, a few mms lower (Gallavardin's sign), a sign characteristic of pulsus alternans, provided alternating extrasystoles (pulsus bigeminus) can be excluded

Although pulsus paradoxus may be appreciated at the wrist, it is more easily detected with the aid of the blood pressure apparatus, by listening to the radial artery at the elbow, during extremely slow deflation of the cuff

*Disparity of readings between the arms* The blood pressure should always be recorded bilaterally. A difference of up to 10 mm is within normal limits. A marked difference of pressure in the two arms (over 15 mm Hg) suggests the possibility of aortic aneurysm, syphilitic or atheromatous dilatation of the aorta, some vascular anomaly, a new growth pressing on the innominate or subclavian artery, thrombosis or embolism of the brachial, axillary or subclavian artery, aortic arch syndrome or pulseless disease, coarctation of aorta, scalenus anticus syndrome, cervical rib, dissecting aneurysm of the aorta, a rigid or sclerotic brachial artery or a physiological (but exaggerated) difference of blood pressure between the two arms,

*Relative brachial or crural hypertension* In all cases of high blood pressure, especially in young individuals, the crural pressure must be recorded to exclude coarctation of the aorta, the pressures in this condition being much higher in the arm than in the leg. In aortic regurgitation, the crural pressure is much higher than the brachial (Hill's sign), it may be 60-100 mm Hg or more.

The mechanism for this exaggeration of the physiologic excess of blood pressure in the femoral artery over that in the brachial is as follows: while the femoral artery is in a direct line with the aorta, the brachial artery comes off from the aorta at a right angle. The femoral artery, therefore, receives blood from the aortic stream at a greater velocity. Since in aortic incompetence the velocity is considerable, there is a pronounced excess of pressure on the femoral artery as compared to the brachial.

**BASIC DEFINITIONS** The difference between the systolic and diastolic pressure is known as the pulse pressure. It is normally 30 to 60 mm (average 40 mm). The mean effective pressure approximates to the diastolic pressure plus one-third of the pulse pressure, it is sometimes referred to as the "average dynamic pressure". Blood pressure readings taken at random and without previous preparation of the patient constitute the so-called casual blood pressure, such readings are usually too high. The basal blood pressure, or the pressure after elimination of all physical, mental and emotional factors, is recorded about ten to twelve hours after a meal, and with the patient at rest and "emotionally desensitized" by resort to frequent recording of the pressures. The static blood pressure (usually about 15 to 20 mm Hg) is the vascular pressure at death or during bouts of cardiac cessation, it is the same in all vessels.

*Normal blood pressures* The arterial blood pressure (brachial) in the normal adult ranges from 95 to 150 mm Hg systolic and 60 to 90 mm diastolic. The normal pulse pressure ranges from 30 to 60 mm. In children, the figures are lower, averaging about 55-35 at birth, 90-60 in early childhood and about 105-65 during adolescence.

Blood pressure is a variable entity, dependent on many factors, such as age, sex, weight, race, climate, familial predisposition, exercise, emotion, nervous tension, eating, smoking and time of day

The *diastolic pressure* is more important than the systolic because (1) it is less affected by extraneous and other influences, and (2) because it reflects the "constant load" or "strain" to which the arterial tree is subject. A rise of diastolic pressure is therefore more significant and harmful than a high systolic level. A fall of diastolic pressure suggests either peripheral vasodilatation with decreased peripheral resistance or aortic regurgitation. A fall of systolic pressure may be indicative of diminution in the strength of contraction of the left ventricle.

There is no unanimity of opinion, as yet, as to what constitutes normal blood pressure, the standards of normality as defined by different observers being a great variance. The common belief that the systolic pressure equals "100 plus age" is grossly inaccurate.

**HYPERTENSION** A blood pressure reading consistently over 140 mm or 150 mm systolic and 90 mm diastolic (provided exertion and emotional factors can be excluded) is abnormal and indicative of high blood pressure.

A rise of both systolic and diastolic pressures is referred to as true hypertension, an isolated rise of systolic pressure as systolic hypertension, and an isolated or preponderant rise of diastolic pressure as diastolic hypertension. Diastolic hypertension is much more significant clinically than systolic hypertension.

Significantly high blood pressure may be due to essential hypertension (also called primary hypertension) which may be benign or malignant, according to the rate of progression and severity of the diseases or secondary to vascular, kidney or endocrine disease, raised intracranial pressure, or polycythaemia vera.

**HYPOTENSION** A blood pressure reading consistently below 90 mm systolic and 50 to 60 mm diastolic is referred to as low blood pressure or hypotension.

Hypotension may be (1) constitutional, (2) acute and transitory, or (3) chronic and persistent.

Some normal healthy individuals display hypotension as a constitutional or familial peculiarity. This type of low pressure, apart from occasional mild symptoms like tiredness, depression and dizziness, is essentially benign.

*Acute hypotension* or a sudden drop of blood pressure, which may or may not carry an ominous significance, may be due to (1) vasovagal syndrome ("common faint"), (2) orthostatic or postural hypotension, (3) carotid sinus reflex, (4) peripheral circulatory failure (or "shock"), (5) acute myocardial insufficiency or cardiac failure, (6) a rapidly developing pericardial effusion, or (7) use of a vasodilator drug like nitrite.

Chronic or persistent hypotension is common in Addison's disease, Simmond's disease, chronic pulmonary tuberculosis, malignancy, anaemia, myxoedema and aortic regurgitation (where it is of diastolic type).

**PULSE PRESSURE.** An abnormal increase of pulse pressure, due to rise of systolic or fall of diastolic pressure, may be encountered in (1) essential and other varieties of hypertension, (2) aortic regurgitation, (3) thyrotoxicosis, (4) fever, (5) complete heart block, or (6) during exercise or emotion, (7) severe anaemias, (8) other hyperdynamic circulatory states, such as

berri-berri, Paget's disease of bone and hepatic insufficiency, (9) arteriovenous fistulae or shunts (e.g. systemic or coronary arteriovenous fistulae, patent ductus, aorto-septal defect and rupture of aortic aneurysm into right heart). The commonest causes of "wide" pulse pressures are aortic regurgitation, atherosclerosis of aorta, thyrotoxicosis and complete heart block. With the exception of the atheromatous or senile aorta (where loss of elasticity of the vessel wall is involved), most examples of wide pulse pressure are due to increased cardiac (or left ventricular stroke) output, with or without associated features, e.g. excessively slow heart-rate and massive regurgitation of blood with ineffective ventricular output.

An excessively *narrow* (or small) pulse pressure of 20 mm or less may be due to (1) the state of "shock" (as encountered after severe bleeding, burns, myocardial infarction, septicaemia, surgical operations and pulmonary embolism), (2) aortic stenosis, (3) cardiac tamponade and constrictive pericarditis, (4) severe paroxysmal tachycardia, (5) tight mitral stenosis with pulmonary hypertension, and (6) advanced stage of congestive heart failure (where the venous pressure is high instead of low, as in the "shock" state). All the aforementioned conditions are associated with decreased cardiac stroke output.

## PRECORDIUM INSPECTION

According to some authors, the first two methods of physical examination, inspection and palpation, can be suitably combined or considered together, since some of the physical signs (such as the apex beat) are perceived simultaneously by the visual and tactile senses. In order, however, to maintain a systematic presentation of the subject, it is preferred to deal with the two methods separately.

**METHOD** For inspection, the patient must be stripped to the waist and examined in a good light, both in the upright (sitting or standing) and in the lying-down (supine) position. The examiner should sit or stand directly facing the patient. A *tangential inspection* of the chest, with the patient recumbent, either from one or other side (Fig. 9.1) or from the foot of the bed may yield valuable information, particularly about pulsations and swellings.

During inspection, the following features are to be noted: (1) the size, shape and type of chest, (2) the shape of the precordium, (3) the position, extent and character of the apical thrust, (4) the presence of other precordial pulsations, (5) the presence of other pulsations of the chest wall outside the precordial area, (6) the presence of extrathoracic pulsations, such as pulsations of the neck and epigastrium, (7) local prominences or bulges of the chest wall, and (8) distended blood vessels over the chest or neck.

**Size, shape and type of chest.** This will be adequately dealt with in connection with the respiratory system. The size, shape and type of chest may have a direct bearing on the presence of atypical or abnormal physical signs in the chest, as in the case of funnel chest, rachitic chest and scoliosis, or the

straight back syndrome where the normal concavity of the vertebral column in the upper dorsal region is absent. It may also be responsible for the diseased condition of the heart, as in emphysema and severe kyphoscoliosis.

**Shape of the precordium.** Normally, the two sides of the chest are symmetrical. Bulging or retraction of the precordium may be due to diseases outside the heart, they should therefore be ruled out first, before incriminating the heart as the cause of abnormality of the precordium.

**PRECORDIAL BULGING** A good sign for recognizing precordial bulge is lateral displacement and elevation of the left nipple in comparison with the right. Bulging or *prominence* of the precordium may be due to (1) Skeletal deformities, such as scoliosis, kyphoscoliosis or rickety deformity of the chest. The presence of scoliotic deformity may be suggested by an asymmetry in the level of the shoulders or of the nipples on the two sides. (2) Disease of the lungs and pleura, such as bronchial carcinoma or pleural effusion, or of the mediastinum, such as mediastinal new growth. (3) Diseases of the heart or pericardium, such as ventricular hypertrophy or pericardial effusion. It may be noted that whilst bulging of the intercostal spaces without involvement of ribs is suggestive of pericardial effusion, bulging of both interspaces and ribs suggests long-standing heart disease, probably originating in childhood or at a time when the bones are soft and incompletely ossified. Bony bulging of the precordium is usually due to mitral valve disease or congenital heart disease. In coarctation of the aorta the upper part of the body is well developed in contrast to the thin legs. Localized bulging of part of the precordial area can rarely be due to a cardiac or ventricular aneurysm secondary to myocardial infarction. (Fig 9 2)

**BACKWARD BULGE** A depressed sternum (pectus excavatum) may give rise to physical signs in relation to the heart depending on the degree of depression or backward bulge. The apex beat tends to be displaced to the left.

**PRECORDIAL FLATTENING** Flattening or retraction of the precordium may be due to cardiac or extra-cardiac causes, the commonest causes being (1) old pericarditis or adherent pericardium, (2) fibrosis or collapse of the lung, (3) scoliosis or kyphoscoliosis. This may also alter the position of the apex thrust.

**The apical thrust** Inspection of the apex impulse is best done by tangential inspection of the chest wall, in a good light, with the patient sitting up and bent slightly forwards or lying down. The apical thrust is a circumscribed systolic elevation, an outward or forward movement of the chest wall, due to the systolic impact of the para- or juxta-apical area of the heart. It is important to note that this thrust is due not to the apex proper but to a small part of the left ventricle above and internal to the apex (juxta-apical thrust). The term cardiac impulse frequently used for this thrust appears unsuitable as it tends to include (besides the apical thrust) all pulsatory phenomena of the chest wall caused by cardiac systole.

**NORMAL APICAL THRUST** In health, the apical thrust is located in the fifth left intercostal space, one-half to one centimetre inside the left mid-clavicular line (Fig 9 3) It is usually three and a half inches (or seven to nine centimetres) lateral to the midsternal line, a distance to be measured not circumferentially but tangentially The midclavicular line (not the mammary or nipple line) is a simpler and more reliable landmark than the midsternal line for describing the site of the apical thrust The normal apex thrust is limited or circumscribed to one interspace, being less than an inch in diameter

**NORMAL VARIATIONS** In infancy or childhood, the apical thrust may be located in the fourth left interspace In thin, narrow-chested and elderly subjects, it may be seen in the sixth interspace in a more medial position than normal In obesity, abdominal distension and during pregnancy, it may be displaced slightly outward and upward by the raised diaphragm

The apical thrust is *invisible* in a fair percentage of healthy adults, particularly when recumbent Absence of the thrust therefore may be due to (1) the heart being situated behind a rib, (2) thick chest wall, (3) pendulous breast, or (3) emphysematous chest In some of these cases, the circumscribed normal thrust is replaced by a diffuse weak impulse When absent in recumbency, the thrust may be rendered visible by making the patient assume the sitting-up posture In children and thin-chested individuals with overacting hearts or after exercise or excitement, the normal apical thrust may appear unduly prominent and diffuse or may even move towards the left with each heart beat

**POSTURAL SHIFT** In a normal heart, the apical thrust displays considerable shift or change of location with alterations of bodily posture Mere turning in bed from the left lateral to the right lateral position may shift the apex as much as one and a half to two inches A change from the recumbent to the upright position or even the taking of a deep breath may appreciably alter the position of the thrust Failure of the apical thrust to shift in this manner (with change of posture or on inspiration) is a sign suggestive of adherent pericardium

**ABNORMALITIES OF APICAL THRUST** These are best determined by palpation However, the apical thrust must be observed for the following (1) presence or absence, (2) location, whether normal or displaced, (3) extent, whether localized or diffuse, (4) direction of movement during systole, whether outward or inward (thrust or retraction), (5) lack of mobility or fixation, and (6) other characteristics

Quite apart from normal variations, the apical thrust may display abnormalities of position, extent or character of diagnostic value, in various diseases Its absence is also significant, especially in the young

**DISPLACEMENT OF THE APEX THRUST** An abnormal shift or displacement of the thrust may be due to extrinsic or intrinsic causes



Extrinsic or extracardiac causes may result in the heart being pushed or pulled from the side, or displaced upwards or downwards by pressure from below or above. Extrinsic causes may be (1) extrathoracic, such as scoliosis, kyphoscoliosis or funnel chest, (2) intrathoracic, such as pleural effusion or pneumothorax, which displace the apex thrust sideways, and aortic aneurysm or mediastinal newgrowth, which push it downwards, or (3) intra-abdominal, such as ascites, meteorism, massive abdominal tumour or advanced pregnancy.

Intrinsic or cardiac causes of displacement include various affections of the heart itself, the most important being congenital disease, such as dextrocardia, where the apical thrust is located on the right side instead of the left, and acquired disease, such as high blood pressure, aortic and mitral valve disease.

Apical shifts of cardiac origin are usually due to hypertrophy or dilatation of one or both ventricles. In left ventricular enlargement, especially hypertrophy, the apex thrust is usually displaced downwards and outwards, in aortic incompetence, it may be located in the sixth or seventh left interspace far out in the axilla. In right ventricular enlargement, the apex thrust is usually shifted outward and is often diffuse.

**EXTENT OF THE APICAL THRUST** The normal apex thrust is less than an inch in diameter and occupies not more than one interspace. A diffuseness of the thrust may be due to (1) thin chest wall, (2) retraction of lung from fibrosis or collapse, (3) overaction of heart from exercise, emotion, fever or thyrotoxicosis, or (4) hypertrophy or dilatation of heart. Whilst in hypertrophy the diffuse thrust is heaving and forcible, in dilatation it is feeble, flapping, tap-like or absent. It is important to note that a diffuse thrust may arise in a forcibly contracting hypertrophied heart, as in aortic regurgitation, as well as in a weak, flabby or dilated heart, even the amplitude of thrust may be similar in the two instances. (5) In ventricular aneurysm and during the acute phase of myocardial infarction, the apex beat may appear diffuse, particularly in a medial and upward direction.

**FORCE OF APEX THRUST** The force of the thrust may be visibly increased in case of (1) thin chest wall, (2) retracted lung, (3) overacting heart, as in thyrotoxicosis, fever or during exertion, and (4) left ventricular hypertrophy, as in hypertension or aortic regurgitation.

In left ventricular hypertrophy, the visibly forcible apex thrust may be accompanied by a mild indrawing of the adjacent midprecordial area, giving rise to a "rocking" or "see-saw" movement of the precordium, the left ventricular rock.

Since palpation alone can decide decisively about the force of the apex thrust, *no deductions must be drawn about force from inspection alone*.

**ABSENCE OF APICAL THRUST** The cardiac causes of an invisible thrust are (1) weak action of the heart, as in myocardial infarction or acute myocarditis,

(2) pericardial effusion, and rarely (3) dilatation of the heart

**Characteristic types of apex impulse** The apex impulse on inspection, may be characteristic enough to permit its immediate recognition and significance. The following types of apex thrusts are noteworthy in this regard.

**NEGATIVE CARDIAC IMPULSE (Škoda's sign)** It is a sucking-in or retraction during systole of the apical region, erroneously regarded in the past as a sign of adhesive pericarditis. It may be due to (1) overacting heart, with apex situated behind a rib, (2) hypertrophied right ventricle, with forward thrust in the midprecordial area and retraction of the apex, and rarely, (3) adhesive pericarditis, a diagnosis only justified when retraction involves both ribs and interspaces, (Broadbent's sign).

**ROUND OR DOME-LIKE APEX IMPULSE** A characteristic, slow-rising, prominent and rounded apex impulse, considered typical of aortic regurgitation, is sometimes recognizable on inspection.

**BIFID IMPULSE** A bifid or double thrust, with two consecutive thrusts of the apical area during each heart beat (visible apical reduplication), may be due to (1) bundle branch block with both thrusts ventricular in origin, or (2) presystolic gallop rhythm with one atrial and one ventricular thrust.

**Other pulsations of the precordium** These are best observed by tangential inspection of the precordial area, preferably with the patient recumbent and the examiner lowering himself sufficiently to bring his eyes level with the anterior wall of the patient's chest (Fig 9 1).

**PHYSIOLOGICAL DIFFUSE PULSATION** In healthy, thin-chested individuals, overacting or hyperdynamic hearts, during fever or after exercise, or with retracted lungs, a diffuse but mild pulsatory movement of the precordium, involving several interspaces, may be noted independently of the apex thrust. It has been described as a *wavy* or *peristaltic* cardiac impulse.

**PHYSIOLOGICAL PARA-APICAL RETRACTION** In thin healthy subjects, a systolic retraction of the chest wall between the apical region and sternum, due to the sucking-in effect of right ventricular systole, may be observed and mistaken for an abnormal apex thrust. It is, however, situated medial to the true apex and is a retraction rather than an outward movement of the chest wall.

**LEFT PARASTERNAL PULSATION** A systolic heaving of the midprecordial area, maximal between the third and sixth ribs, and frequently involving the lower half of the sternal bone, is characteristic of massive right ventricular hypertrophy. A central lift may also be due to systolic expansion of left atrium from mitral regurgitation. If there is gross dilatation of this chamber there may also be some pulsations to the right of the sternum and a rocking motion of the entire chest wall. In left ventricular hypertrophy rotation of the septum may be so pronounced that the left ventricle may come to lie beneath the left anterior chest wall. A heave in the left parasternal region may be due to left ventricular hypertrophy. The clue is the absence of pulsation in the epigastrium. A pericardial effusion sometimes gives rise to a curious undulating appearance in the intercostal spaces to the left of the sternum.

**ROCKING OR SEESAW MOVEMENTS** of the precordium may be observed in massive hypertrophy of the right or left ventricle. In *right ventricular* hypertrophy, an inward movement of the apex is associated with an outward movement of the midprecordium during systole, in *left ventricular* hypertrophy, the phenomenon is reversed.

**DIFFUSE SYSTOLIC RETRACTION** A diffuse retraction of the precordial area, involving ribs and interspaces, may be due to (1) tricuspid regurgitation, (2) adhesive pericarditis or (3) aortic incompetence.

**TRANSITORY PRECORDIAL OR PARA-APICAL PULSATION** A local area of pulsation, medial to and above the apex thrust, may appear transitorily, for a few days, in case of recent myocardial infarction.

**HERZSTOSS** A massive outward pulsation of the entire or major part of the precordium during systole, usually associated with massive hypertrophy of the heart in a thin-chested individual.

**Other pulsations of the chest wall** **HIGH THORACIC PULSATIONS** Pulsations of the upper part of the chest, often better seen than felt, are usually indicative of disease. When observed in the second right interspace or behind the upper part of the sternum (upper sternal pulsation), they are indicative of (1) aneurysm of the ascending or transverse part of the aortic arch, (2) dilatation of the aorta, or (3) aortic regurgitation. In the later stages of an aneurysm, a rapidly growing, massive prominence or bulge (Fig 9 4) with expansile pulsation may appear in the same area and eventually lead to fatal termination from external haemorrhage.

Pulsations involving the second or third left interspace may be due to (1) dilatation of the pulmonary artery or conus, as in case of patent ductus arteriosus, and septal defect, mitral stenosis or aneurysmal dilatation of pulmonary artery, (2) retraction of the left lung from fibrosis or collapse, and rarely (3) aneurysm of the descending thoracic aorta.

**MASSIVE PULSATORY THRUSTS OF THE CHEST WALL** These may be observed in cases of massive cardiac hypertrophy. In massive *left ventricular* hypertrophy, the left thoracic wall may move outward and the right wall inward during systole, the movements being reversed in diastole. In massive *right ventricular* hypertrophy, the anterior thoracic wall may be pushed forward and the lateral walls inward during systole. In massive enlargement of the *right atrium*, as in advanced tricuspid valve disease, the right lateral wall may be pushed outward during systole.

**LOCALIZED NEGATIVE PULSATIONS** This sign refers to a systolic indrawing or retraction of the tenth and eleventh interspaces, on the left side, in the scapular or posterior axillary line. Though considered diagnostic of adhesive pericarditis, it may occur also in the case of cardiomegaly or an unduly thin chest in a normal individual, although if the ribs themselves are pulled in with systole (systolic retraction) the diagnosis of adhesive pericarditis is confirmed (Broadbent's sign).

**PULSATIONS OF STERNOCLAVICULAR JOINT AND STERNUM** (1) Pulsations of the right sternoclavicular joint may suggest a right sided aortic arch (2) Pulsation of either sternoclavicular joint occurs in aortic dissection or aneurysm (3) Systolic outward pulsation of upper half of sternum is generally due to aneurysm of the ascending aorta

Pulsation to the right of sternum may result from a dilated and unfolded or frankly aneurysmal ascending thoracic aorta, rarely it may be due to a large right atrium

**PULSATIONS IN ATYPICAL SITUATIONS** These may be observed anywhere on the chest wall, including the back. These are observed in the case of (1) empyema necessitatis, a local pulsatile swelling over the lateral aspect of the chest wall in case of empyema, (2) highly vascular tumour, such as lymphosarcoma, (3) aneurysm of the descending thoracic aorta, in the back (Fig 9 5), (4) aneurysm of the innominate artery (Fig 9 6), in the supraclavicular region or upper part of thorax, and (5) coarctation of the aorta, with prominent and pulsating vessels in the interscapular and intercostal regions, best seen with the patient bent forwards and arms hanging down (Suzman's sign)

**Extrathoracic pulsations** Pulsations of the suprasternal notch, carotid arteries, jugular veins and epigastrium must be routinely looked for during inspection, as they may afford information of diagnostic value

**SUPRASTERNAL OR EPISTERNAL PULSATION** (Fig 9 3) A pulsation in the suprasternal notch is a fairly common finding, even in apparently normal subjects. It may be seen in health if patient is elderly and kyphotic or may be due to (1) overacting or hyperdynamic heart, (2) anaemia, (3) aneurysm of the aorta, (4) dilatation of the aorta, as in atheroma or syphilitic aortitis, (5) raised or uncoiled aorta, as in hypertension, (6) elongation and flexion of the innominate artery, (7) anomalous right subclavian artery, or (8) thyroidea ima artery

**Pulsations of the vessels.**

**Jugular venous pulse.** Observation of the form of the venous pulse and the level of venous distension can supply important information

Engorgement and visible pulsation of neck veins may be observed even normally in the recumbent position, but are certainly abnormal when observed above the level of the manubrium sterni in the sitting up or semiproped (45° from horizontal) posture. They are indicative of a raised jugular venous pressure (JVP). Veins observed, as a rule, in the neck, or either side are (1) the external jugular vein, which is quite superficial and therefore clearly observed, and (2) the deep-seated internal jugular vein, best seen as a pulsation beneath the sternocleidomastoid muscle

**The normal jugular venous pulse** Pulsation in the superficial jugular veins and over the jugular bulb is a normal phenomenon. The venous waves are most accurately reproduced in the internal jugular vein which is in direct line with the right atrium. In order to observe the jugular pulse, the patient

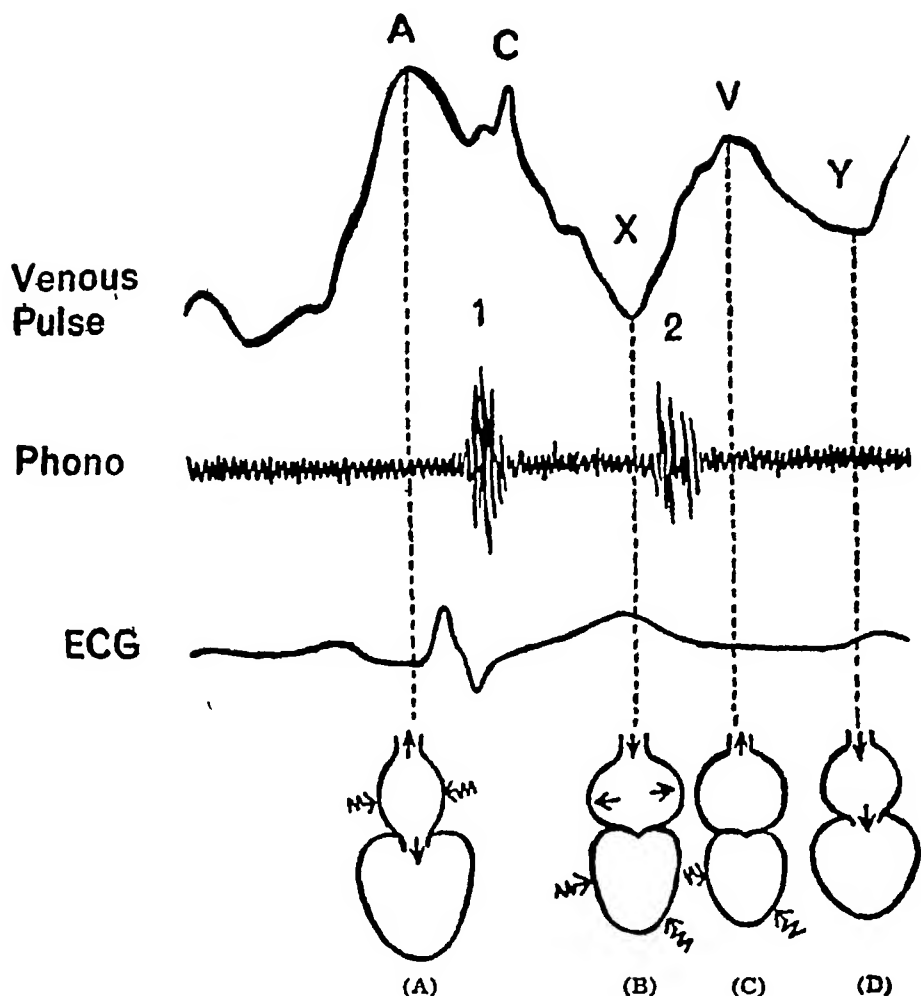


Fig.9.7 Relationship of electrocardiogram, jugular venous tracing and phonocardiogram to the haemodynamics of the right heart (A) Atrium contracting, valve open and a wave (B) Atrium relaxed, filling, valve closed and x trough (C) Atrium tense, filled, valve closed and v wave (D) Atrium emptying, valve open and y trough The c wave is an artifact transmitted from the carotid pulse

should be lying comfortably either flat or with a pillow under the head. The examiner should look carefully at the lower neck of the side under inspection just lateral to the clavicular head of the sternomastoid muscle for the double outward pulsation of the normal venous pulse. Since the pulsations are outward, a convenient method of observing the venous pulse is to look at the patient full face, with the head of the patient in the midline position, and simultaneously palpating the opposite carotid artery with the thumb

Absence of pulsation may mean either that the vein is fully distended and the top of the column of blood lies within the head, or the vein is collapsed and the top of the column lies within the chest. To find this out, press a finger lightly over the lower end of the jugular vein. If the vein fills, it suggests that the venous pressure is too low to be recorded and the jugular pulse may be assumed to be normal. If the vein is not rendered visible due to elevated venous pressure, the patient should be made to sit up when the top of the column of blood may become visible.

Venous pulsation in the neck is the only clinical method available for determining atrial activity. It is not due to the speed of a pulse wave, as in the case of arterial pulsation, but to an alternate filling and emptying of the vein with blood, caused by pressure and volume changes within the atrium. Venous pulsations must be distinguished from pulsations of the carotid arteries.

The venous pulse is due to variations in the volume of the veins of the neck caused by a greater or lesser filling, which is dependent on the oscillations of right atrial pressure during the cardiac cycle (Fig 9.7). It consists of three main waves or crests, A, C and V, the summits of which are presystolic, systolic and diastolic in time, and two negative waves or troughs, X and Y.

**DIFFERENTIATION OF VENOUS FROM ARTERIAL PULSATION** Since the internal jugular vein lies deep to the sternomastoid muscle, its pulsations are often confused with those of the common carotid artery. A striking pulsation in the neck, when associated with small pulse at the wrist, almost always suggests a venous rather than an arterial pulsation. Similarly the presence of marked venous engorgement elsewhere in the body favours neck pulsation as being venous. The following table gives the essential differences between venous and arterial pulsations.

	JUGULAR VENOUS PULSATION	CAROTID ARTERIAL PULSATION
1 <i>Position</i>	Pulsation more lateral	Pulsation more medial
2 <i>Visible or palpable</i>	Better seen than felt	Better felt than seen
3 <i>Wave form</i>	Multiple gentle pulse waves	Single brief pulsation
4 <i>Timing</i>	The first venous wave precedes the carotid pulse upstroke, the second wave follows the carotid pulse	Coincides with ventricular systole
5 <i>Effect of compression of vessel</i>	Light pressure over the vein at the root of the neck will eliminate the pulsation	Much greater pressure over the artery is required to eliminate the pulse
6 <i>Effects of respiration, posture and abdominal compression</i>	Venous pulse is decreased with inspiration or sitting up, and exaggerated with expiration, lying down or abdominal compression	Unaffected
7 <i>Valsalva manoeuvre</i>	Distension of neck vein increases	No effect

**Abnormal jugular pulsation** Exaggeration of the A wave is the commonest abnormality of the venous pulse. The A wave in the right jugular vein can be identified by palpation of the left carotid artery. The A wave just precedes the carotid pulse. Alternatively the examiner can auscultate the heart while looking at the jugular venous pulse, the first sound occurs on the descending limb of the A wave. Common alterations in the jugular venous pulsations with their clinical significance are presented in a tabulated form below.

ABNORMAL VENOUS WAVE	CLINICAL SIGNIFICANCE
<b>A WAVE</b>	
<i>Absent</i>	Declines in right heart failure, disappears with atrial fibrillation, and A-V junctional rhythm (some cases)
<i>Fused (with C wave)</i>	Supraventricular tachycardia
<i>Exaggerated</i> (Giant A wave, venous Corrigan pulse)	(1) Due to right atrial contraction against increased resistance tricuspid stenosis or atresia (2) Abnormal compliance of right ventricle Pulmonary stenosis, pulmonary hypertension Right atrial tumor or thrombus Ebstein's disease (3) Hypertrophic obstructive cardiomyopathy (4) Valvular aortic stenosis (sometimes) (5) Pericardial effusion, constrictive pericarditis, restrictive cardiomyopathy
<i>Cannon waves</i> (Special form of giant A wave in early systole)	Occur whenever right atrium contracts against a closed tricuspid valve (1) Isolated Ventricular ectopics (2) Regular (a) At normal heart rate Nodal rhythm/ sinus rhythm with long P-R interval (b) At rapid heart rate Supraventricular tachycardia especially nodal (3) Irregular (a) Slow rate Complete heart block (b) Rapid rate Ventricular paroxysmal tachycardia
<i>Non conducted</i>	Atrial tachycardia or atrial flutter with block
<b>X WAVE</b>	
<i>Absent</i>	Tricuspid regurgitation
<i>Large</i>	Constrictive pericarditis, restrictive cardiomyopathy, atrial septal defect (sometimes)
<b>V WAVE</b>	
<i>Large (Fused CV waves, regurgitant wave)</i>	Tricuspid regurgitation
<b>Y WAVE</b>	
<i>Slow descent</i>	Tricuspid stenosis
<i>Prominent rapid descent</i> (due to sudden release of blood damped back in left atrium during ventricular systole)	Preceded by regurgitant systolic wave Tricuspid regurgitation Not preceded by regurgitant systolic wave Constrictive pericarditis (Friedreich's sign, diastolic dip), restrictive cardiomyopathy, severe heart failure

**Jugular venous pressure (JVP)** The sternal angle is the most widely used and satisfactory point from which to measure the jugular venous pressure,

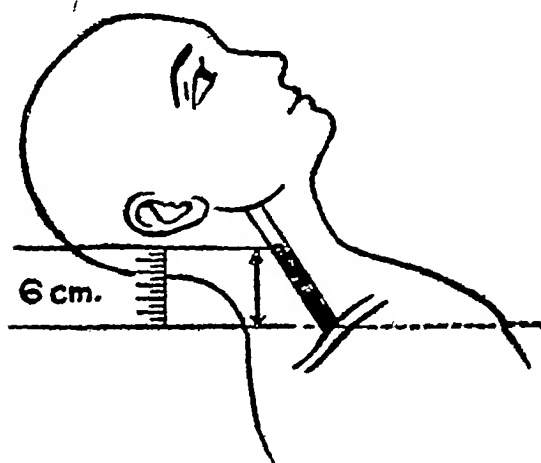


Fig 9 8 Clinical assessment of jugular venous pressure

since it roughly corresponds to the level of the mid-right atrium and represents approximately the normal pressure, whether the body is horizontal or erect or in any intermediate position. The pressure is expressed in terms of centimetres for the vertical distance between the top of the column of blood and the sternal angle during quiet respiration (Fig 9 8). For clinical estimation of jugular venous pressure the patient must be propped up at an angle of  $45^\circ$  with the head in a relaxed position since the internal jugular vein is situated

deep to the sternomastoid muscle. Rotation of the head slightly away from the side under observation may help to make the vein more prominent. The level to which the internal jugular vein is distended, or the height to which the pulsations ascend is the actual height of venous pressure. If the pulsations cannot be seen at  $45^\circ$ , the head should be lowered to  $30^\circ$ . If the veins appear distended when the patient is in a horizontal position, but are collapsed and not visible at  $30^\circ$  elevation, the venous pressure can be said to be normal. If the venous pressure is very high, the patient has to be propped up in a sitting position. The upper limits of normal pressure at various degrees of head and trunk elevation are recumbent + 2 cm,  $30^\circ$  + 3 cm,  $45^\circ$  + 4.5 cm, and upright level of suprasternal notch.

If the upper level of the pulsation is not visible even in the sitting position, venous pulsations can be seen behind the angle of the jaw and may be transmitted even to the ear lobes.

It is necessary to examine the veins on both sides of the neck. Occasionally only the left jugular vein is seen to be distended. This may be seen especially in older subjects with atherosclerosis due to pressure of the dilated aortic arch on the left innominate vein.

The external jugular veins are less reliable for estimation of venous pressure because (a) They possess venous valves which are not found in the internal jugular veins. The level to which the internal jugular vein is distended is therefore the right atrial pressure. (b) The external jugular vein shows delayed appearance of the wave form because of narrow vessel diameter. (c) The external jugular veins are prone to undergo kinking, and may give a falsely high value of venous pressure. (d) In presence of severe cardiac failure, a low pressure reading may be obtained because of a Venturi effect where the vein joins a larger vessel through which there is rapid blood flow.



*Gaertner's method* utilizes a prominent antecubital or superficial hand vein for the measurement of venous pressure. The arm is raised, with the patient recumbent until the vein collapses. The height of the collapsed vein above the level of the right auricle represents venous pressure. Visible engorgement of the veins over the undersurface of the tongue, in the sitting up posture, is also evidence of a raised venous pressure (May's sign).

*Causes of elevated venous pressure* (1) Jugular venous hypertension (Fig 9.9) is the earliest evidence of general systemic congestion in cardiac failure. (2) Obstruction or pressure on the superior vena cava of an aortic aneurysm, mediastinal tumour or lymphoma. While venous engorgement of heart failure is associated with oedema of legs and hepatomegaly, that of superior vena caval origin is associated with visible distension of collateral veins over the upper part of the chest. (3) Hyperkinetic circulatory states due to fever, pregnancy, anaemia, arteriovenous aneurysm, thyrotoxicosis or advanced disease of the liver, increased blood volume as in pregnancy and acute nephritis or large intravenous infusions, bradycardia, increased intrathoracic, intra-abdominal or intrapericardial pressure, and tricuspid stenosis. (4) Reduced volume capacity of R V. Pericardial effusion or constrictive pericarditis, endomyocardial fibrosis, right ventricular tumor, bulging of ventricular septum (Bernheim effect), and obstructive cardiomyopathy. (5) Increased blood volume especially when resulting acutely as in overhydration with infusions, acute nephritis, steroid therapy. (6) Raised intra-pleural and intra-abdominal pressure—large pleural effusion, tight abdominal binders, pregnancy.

*Transient engorgement* or distension of neck veins, indicative of raised venous pressure, may be due to physiological causes, such as exercise, coughing, expiration and raised intra-abdominal pressure from a tight belt or binder.

*Persistent unilateral engorgement* of the jugular vein in the absence of dyspnoea is due to impaired venous inflow into the right side of the heart and suggests constrictive pericarditis, tricuspid stenosis or obstruction of superior vena cava. In superior vena caval obstruction, the liver is not congested and the venous pressure is raised only in the upper part of the body. Unilateral distension of the neck vein may be due to local obstruction or pressure on a vein or to the syndrome of kinked innominate vein, usually secondary to pressure from a high and tortuous aortic arch at the origin of the common carotid artery. The vein should be observed after massaging it to see if it fills from below.

**HEPATO-JUGULAR REFLUX** With the patient breathing normally, firm, sustained pressure is applied over the right upper abdominal quadrant for about one minute (if the liver is tender pressure should be exerted elsewhere over the abdomen). The reflux is positive if the veins get distended and the level of venous pulsation rises more than 1 cm in the neck. The abdominal compression test may reveal latent elevation of venous pressure. It also helps to differentiate venous from arterial pulsation in the neck.

*Inspiratory distension of neck veins (Kussmaul's sign)* is an exaggeration of normal venous filling on inspiration. In health the compliance of the right heart is such that the increased venous return on inspiration is accepted without significant pressure change. In chronic constrictive pericarditis or cardiac tamponade, severe congestive heart failure, or constrictive cardiomyopathy, the right heart compliance is reduced, increased venous return being reflected by increased venous filling of neck veins.

*Expiratory increase in venous pressure* may occur in some cases of bronchial asthma and chronic obstructive pulmonary disease.

### Arterial pulsations

**CAROTID PULSATION** "*Dancing*" carotids. Massive pulsation of the neck arteries, the so-called dance of the arteries, is commonly observed in cases of aortic regurgitation (Corrigan's sign). This may be severe enough to cause visible movement of the ears or head with each beat of the heart (Alfred de Musset's sign).

Visible pulsations of the neck arteries of a lesser order may be seen in cases of hypertension, thyrotoxicosis, anaemia, systemic arteriovenous fistula, coarctation of aorta and in thin and nervous individuals. Pulsating carotids are usually indicative of a wide arterial pulse pressure.

*Absence or diminution of pulsation* of the carotid and subclavian arteries, associated with normal pulsation of the femorals (so-called reversed coarctation) is a characteristic finding of Takayasu's disease or the so-called "pulseless disease". Poor pulsation of one or both carotid arteries may be caused by aortic stenosis or carotid insufficiency. The opposite condition of normal or increased arterial pulsation in the neck with poor or absent femoral pulsation is encountered far more commonly in cases of coarctation of the aorta.

A *kinked carotid artery* may give rise to a small pulsatile oval swelling visible above the right clavicle in females with hypertension, atheroma and kyphoscoliosis. Prominent pulsation on the right side of the neck, in hypertension, is sometimes referred to as Rowntree's sign. A kinked carotid artery in a male is suggestive of coarctation of aorta.

*Carotid swell*. Obstruction of the aorta in coarctation may result in excessive carotid and subclavian pulsation.

**EPIGASTRIC PULSATION**. Pulsation in the epigastrium is common. It is important in diagnosis. Points to note about such a pulsation are (1) whether it is strictly systolic (synchronous with the apex thrust) or delayed (occurring after the thrust), (2) whether this pulsation is a thrust or retraction, (3) whether situated high up and close to the xiphisternum or low down near the umbilicus, and (4) whether central or to the right or left of the midline.

An epigastric pulsation may be cardiac, aortic or hepatic in origin. The *cardiac type* may be due to a hypertrophied right ventricle as in mitral stenosis, or a displaced cardiac apex as in massive left-sided pleural effusion. It occurs synchronously with the apex thrust, is more of a retraction than a thrust, and is located high up in the epigastrium. The *aortic type* may be due to (a) nervousness associated with a thin abdominal wall, (b) normal aortic pulsation transmitted by an abdominal lump or tumour, such as a malignant pylorus or an enlarged liver, or (c) an aneurysm of the abdominal aorta. The aortic type of pulsation occurs soon after the apex thrust, is a forward movement of the abdominal wall and is located low down in the epigastrium. The *hepatic type* of pulsation is rare and due to an enlarged and pulsating liver, as in tricuspid regurgitation or stenosis. It usually occurs soon after the apical thrust, and is associated with a palpably enlarged and pulsating liver.

*Distended subcutaneous arteries* Dilated and tortuous superficial arteries under the skin of the chest and back are a characteristic feature of coarctation of the aorta, and are due to the development of an anastomotic or collateral circulation. The vessels, which display visible and palpable pulsations together with thrills and murmurs, are most obvious in the interscapular and infra-scapular regions of the back, particularly with the patient bending forwards and the arms hanging down by the sides (Suzman's sign).

## PALPATION

### OBJECT

The value of palpation is only second to that of auscultation.

### METHOD

For palpation, the patient, stripped to the waist, is first examined sitting or standing, preferably bent slightly forwards. The apex beat is more easily palpable in this position than in the supine position. The examiner stands or sits on the right of the patient and places the *palm* of his hand (Fig 9 10) which must be warm, on the area under investigation. After palpating with the open hand (palmar palpation) both with light and heavy pressure, further details about localization and character of pulsations are determined with the tip of the index finger or the tips of the second and third fingers of the right hand (digital palpation) (Fig 9 11).

The same procedure is then repeated with the patient recumbent. In special cases, as for instance for determining the mobility of the apex, palpation may be carried out in several different positions. In female patients, it often proves necessary to manually displace the breast upwards or upwards and outwards, for a proper palpation of the apical thrust.

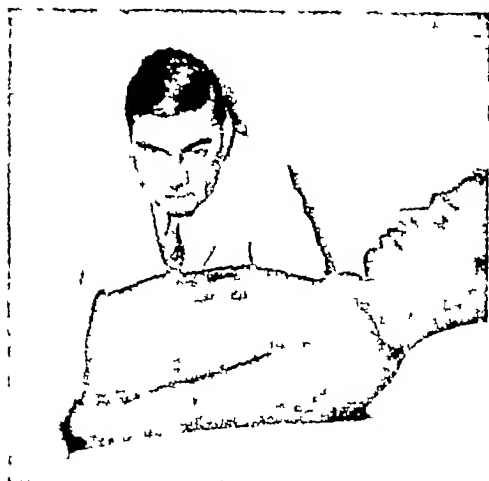


Fig 91 Tangential inspection of precordium

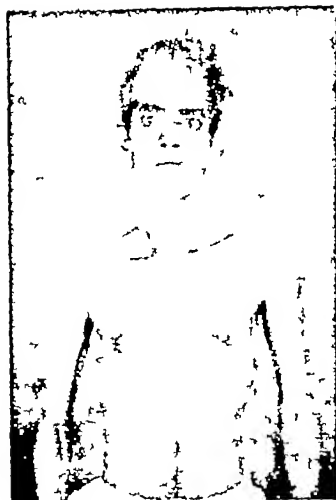


Fig 92 Localized bulging of part of precordium from ventricular aneurysm

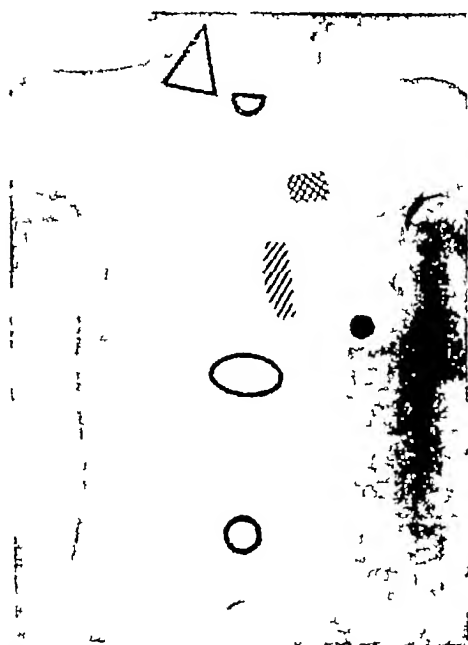


Fig 9.3 Positions of normal apex beat and other common pulsatory phenomena

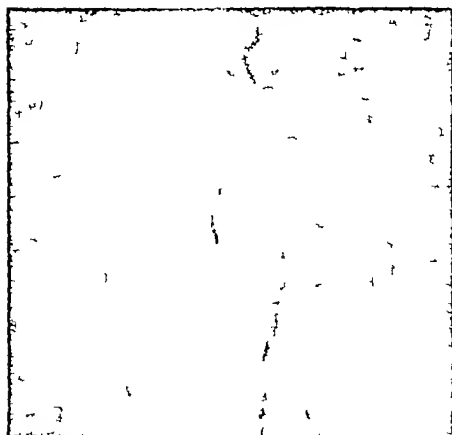


Fig 94 Aneurysm of aorta eroding through the sternum

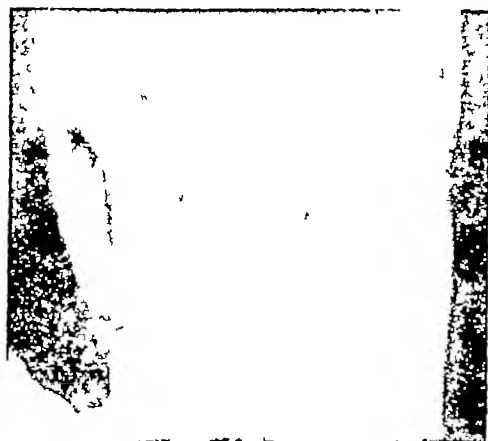


Fig 95 Huge aneurysm of the descending thoracic aorta

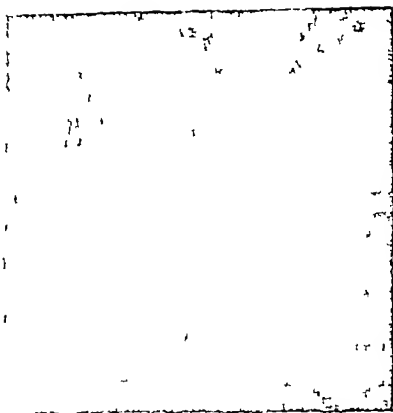


Fig 97 Massive aneurysm of  
subclinate artery

Figs 97 and 98, see pp 216 and 219  
respectively

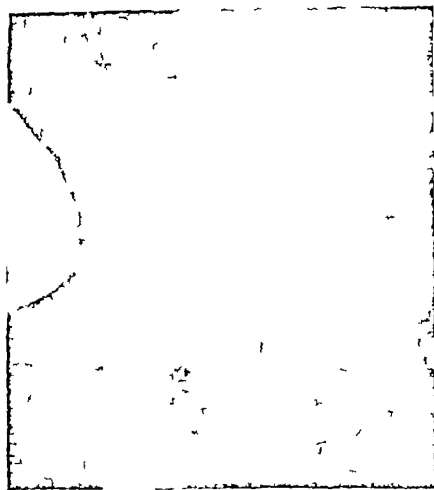


Fig 99 Prominent jugular vein in  
congestive cardiac failure

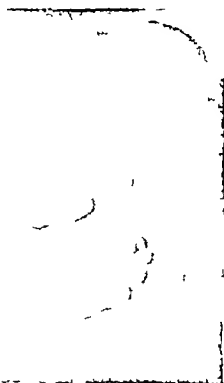


Fig 910 Palmar palpation of  
apex beat

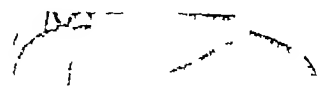


Fig. 911 Digital palpation of  
apex beat.



Fig. 912. Method of detecting  
tracheal tug

Figs 913 to 925 see pp 230 304

## SCHEME OF EXAMINATION

Palpation includes a systematic examination of the following (1) the apex beat and the detection of the character of the cardiac impulse, (2) other pulsations over the chest, neck or epigastrium, (3) thrills, (4) friction rub or fremitus, (5) palpable sounds, and (6) tracheal tug

**Apex beat** **DEFINITION** It has been customary to define the apex beat or apical thrust as the lowermost and outermost point of distinct cardiac pulsation. Such a definition is not entirely satisfactory. In view of the contour of the chest wall and its relation to the heart, an underlying pulsation (such as the pulsation of the apex), when communicated or transmitted to the chest wall, may be appreciable for some distance outside the true or anatomical site of pulsation. The outer border of the communicated pulsation is then likely to be erroneously regarded as true apical pulsation.

The apical impulse has been alternatively defined as the point of maximum impulse or thrust (*P M I* for short). In view of the fact, however, that in certain diseases, such as aortic aneurysm, the point of maximum pulsation may be located well away from the true apex, this definition also appears unsatisfactory.

For these reasons, the *apical thrust* is best defined as the *lowermost and outermost point of the cardiac impulse, which imparts a perpendicular thrust to the palpating finger*. This definition seems to conform most closely to the true position of the cardiac apex.

**FORCE AND CHARACTER OF THE CARDIAC IMPULSE** The apex beat as well as the other pulsations of the chest are usually better felt in children and young individuals than in the middle-aged or elderly. This is because, in the former, the size of the heart in relation to the capacity of the chest is greater, the diaphragm higher and the thickness of the covering lung tissue and integument less. Palpability of the apex impulse is greater in thin-chested and asthenic individuals, especially in those with overacting hearts, than in the fat or the emphysematous.

**Normal apical impulse** The features of the normal apical impulse are (a) It does not exceed half the distance between midsternal and midaxillary lines (b) It occupies only one rib interspace (c) Its outward movement does not persist beyond early systole.

### Abnormal apical impulse.

**Heaving or sustained apical thrust** In left ventricular hypertrophy associated with systolic or pressure overload as in aortic stenosis or systemic hypertension, the apical thrust is a heaving, thrusting, resistant sensation (the palpating finger being lifted slowly). This is because the strain on the ventricle during systole results in a concentric hypertrophy of the left ventricle without much increase in volume.

*Hyperdynamic (forceful) thrust* Such a thrust suggests diastolic or flow overload of the left ventricle as in mitral or aortic incompetence, patent ductus arteriosus or ventricular septal defect. This is because overfilling and distension of the left ventricle occurs in diastole resulting in eccentric hypertrophy, that is, hypertrophy and dilatation with cardiomegaly.

*Snappy or slapping apex beat* In an overacting heart, as in thyrotoxicosis, fevers and after exercise or excitement, especially when associated with a thin chest wall, the apical thrust may become snappy or sudden and forcible. Such a thrust can be distinguished from a heaving apex thrust by its suddenness or rapidity of outgoing movement and by its greater amplitude (hyperdynamic thrust).

*Tapping apex beat* In mitral stenosis, the apex beat is felt as a sharp systolic tap, shock or rap, the palpable equivalent of the first heart sound. This is often described as a short, snappy or hurried apex thrust, or closing snap of the mitral valve.

The "tapping" nature of the apex impulse in mitral stenosis is attributable to two factors. (a) A shortening of the outward systolic thrust of the left ventricular apex, the latter being normally caused by the momentary recoil of the left ventricle against the chest wall during ejection of blood into the aorta. In mitral stenosis, the left ventricle, receiving less blood as it does from the left atrium and not being hypertrophied, the systolic thrust is abbreviated or shortened. (b) The normal slow, outward wave (surge) or movement of the left ventricle during diastole (as it swells with blood) is reduced or absent in mitral stenosis, because of reduced ventricular filling. It is this combination of a short systolic thrust and reduced ventricular filling during diastole that gives the apex impulse its sharp, short and tapping nature in mitral stenosis. In some cases of mitral stenosis, even when "tight" or "severe", the apex beat may appear normal or even exaggerated. This is due either to a massive or aneurysmal left atrium pushing the left ventricle forwards, or to right ventricular dilatation and failure with a hyperdynamic heart. In mitral regurgitation, on the other hand, it is hyperdynamic or heaving and laterally displaced.

*Absent or impalpable apex impulse* Impalpability of the apical impulse may be due to (a) Thick chest wall or obesity (b) An overlying rib (c) Pulmonary conditions such as emphysema (d) Pericardial effusion. A definite apex beat denies the presence of much fluid except in the unusual case where a large effusion is entirely posterior. (f) Constrictive pericarditis (g) Displaced apex beat. Before deciding that the impulse is not palpable, dextrocardia congenital or acquired, or displacement due to thoracic deformity or pulmonary conditions such as pneumothorax or fibrosis of the lower lobe of the right lung should be excluded by palpating on the opposite side.

*Double impulse* A bifid, double or duplicated impulse is better felt than seen. It may be due to (a) *Presystolic gallop*. The forceful atrial contraction needed to fill a failing left ventricle will produce a palpable presystolic

thrust The additional thrust makes the cardiac impulse begin before systole and produces a sensation of hesitation on the rise, in severe cases a distinct double impulse can be felt (b) *Protodiastolic gallop* The extra beat follows closely on the second sound and may be palpable as a diastolic thrust (c) *HOCM* A double impulse in hypertrophic obstructive cardiomyopathy is caused by a large atrial contraction However in some patients with this condition a second late systolic impulse occurs as left ventricle emptying is obstructed in the middle of systole (d) *Constrictive pericarditis* Here a thrust is noticeable in early diastole at the time of the third sound as rapid ventricular filling comes to an end As the usual systolic thrust is reduced in this condition, it may become the main feature of the cardiac impulse (e) *L V aneurysm* Impulses distinctly separate from one another may be produced by a left ventricular aneurysm (f) *Normal* Rarely a normal third heart sound can be felt particularly in pregnancy (g) *Miscellaneous* Double apical thrusts with both impulses occurring in systole, are strongly suggestive of HOCM but they may also occur in some cases of myocardial infarction, angina pectoris, or the systolic click syndrome associated with prolapse of the mitral valve into the left atrium

*Retractile apex impulse* In constrictive pericarditis the apex moves inwards during systole, or there is retraction of the apex because the pericardium is adherent to the chest wall so that decrease in size of the ventricle will be transmitted to the chest wall and the thickened pericardium will not allow the forward movement of the apex

*Paradoxical apex impulse* Outward movement of some part of the precordium, often over the apex of the heart, occurs when there is a ventricular aneurysm which fills and expands during systolic contraction of the ventricle

*Feeble impulse* In serious myocardial disease such as acute myocarditis or myocardial infarction the apex impulse may become diffuse and feeble

*Confluence or fusion of apex impulse* with another area of pulsation over the precordium, thus simulating a medial or upward extension of the apical impulse, may occur as a transient finding in acute myocardial infarction.

*Myotonic impulse* An undue slowness or lag of downward movement during diastole may be noted in ventricular aneurysm of the heart

**MOBILITY** The mobility of the cardiac apex can be assessed by determining its location in various positions of the body, such as the right and left lateral positions, upright and recumbent positions, and during phases of deep breathing

**2 Pulsations at abnormal sites** Other pulsations of the chest, neck and abdomen, already discovered by inspection, are now confirmed and studied in greater detail with the help of palpation Although the amplitude and extent of a pulsation are often better seen than felt, its force and character are better appreciated by palpation



**RIGHT VENTRICULAR "HEAVE" OR "SLAP"** Depending on hypertrophy or overaction of the right ventricle, pulsation in the midprecordial or left parasternal area (best felt with the breath held in expiration) may display either (a) a slow and heaving pulsation in case of hypertrophy (right ventricular heave), or (b) a sudden, forcible or tumultuous pulsation in case of overaction of the heart (right ventricular slap). By observing the duration and quickness of the lift, it may be possible to distinguish clinically between flow and pressure loads of the right ventricle. In the flow load type of impulse e.g. in atrial septal defect (uncomplicated) there is an outward movement of the left parasternal area which is quick and slightly sustained but only during early systole. This outward movement is followed as a rule by a retraction of the parasternal area during the rest of systole. In the pressure load type of impulse (in moderate to severe mitral stenosis) there is a sustained parasternal lift throughout systole. In early or mild cases of mitral stenosis the "slapping" apex beat is associated with little or no right ventricular lift.

**PULSATING LIVER.** An expansile pulsation of the liver, in case of tricuspid regurgitation or stenosis, resulting in one variety of epigastric pulsation, is usually obvious on palpation. The expansile nature of the pulsation can be elicited either by (1) bimanually palpating the liver with one palm held over its anterior surface and the other over its posterolateral surface, or (2) by firm pressure with the two palms over the anterior surfaces of the right and left lobes respectively, or (3) by firm pressure of the closed fist against the lower ribs overlying the anterior surface of the liver.

*Pulsations to the right of the sternum* may be due to an unfolded aorta or aneurysm.

*Arterial pulsations due to enlarged collateral arteries* in coarctation of aorta can be seen and felt particularly in the interscapular regions (best observed when the patient bends forward with the arms dependent), along the ribs, and just lateral to the sternum.

**3 Thrills.** **DEFINITION** A thrill may be defined as a vibratory sensation or tremor originating in the heart or aorta, and transmitted to the palpating hand through the chest wall. A thrill can be compared to the sensation experienced by the hand when placed over the throat of a purring cat, a feature appreciated by Laennec when describing the presystolic thrill of mitral stenosis as "*fremissement cataire*". A thrill is of little diagnostic value, being invariably associated with a loud murmur. In case of a very loud or diffuse murmur, the site of the thrill may serve to determine the point of maximum localization of the murmur. The timing of a murmur within the cardiac cycle may be facilitated by attention to the thrill, whenever present. A thrill without a murmur is usually not a thrill at all, being due to vibration of the chest wall through some other cause (e.g. loud first sound). Diffuse thrills are suggestive of very loud murmurs.

The use of the word thrill has been unfortunately extended to include a host of unrelated phenomena such as the "fluid thrill" of ascites, "hydatid thrill" of hydatid cyst, "vascular thrill" of arteriovenous fistula, "thyroid thrill" of toxic goitre

**MODE OF PRODUCTION** The production of a thrill depends on the passage of blood through a narrowed or stenotic valve leading to the formation of eddies in the blood, thus setting the surrounding structures into vibration

**METHOD OF PALPATION** A thrill may be felt best either with the palm of the hand, the ulnar border of the hand or with the fingers. Thrills are often appreciated best with the heads of the metacarpal bones. They are usually felt better with light than with heavy palpation. In case of doubt a thrill may be rendered more obvious by change of position, exercise which accelerates the flow of blood or forced expiration which brings the heart closer to the chest and retracts the lung. Whilst the thrill of aortic stenosis is usually best felt in the "sitting up and leaning forward" position, that of mitral stenosis is as a rule best appreciated in the left lateral decubitus

**NOTEWORTHY FEATURES** It is worth remembering that thrills are much less common than murmurs, are usually associated with low frequency or harsh murmurs, and are common with stenotic or obstructive rather than regurgitant murmurs. Thrills maximal at the base are usually systolic whilst those at the apex are usually diastolic. In rare cases, a thrill is present without an audible murmur, this is usually due to the latter being of very low pitch.

The shock or tremor imparted to the chest wall by an overacting heart with a thin chest wall may be erroneously diagnosed as a thrill. The diagnosis of a thrill is only justified when it shows (a) a definite sense of vibration, (b) a moderate length or duration, and (c) an accompanying murmur. In the case of the hyperdynamic impulse, the tremor or vibration of the chest which may simulate a thrill is coarse and ill-sustained (better described as an apical or cardiac *shudder* rather than "thrill"). A short systolic thrill may at times be felt above the right clavicle in perfectly healthy individuals.

**CHARACTERISTICS** The main features to study about a thrill are (1) the *site* of maximum palpability, (2) its *extent*, and (3) its *timing* in relation to the cardiac cycle. These determine its significance. The "timing" of a thrill is best carried out with the apical thrust. A thrill that occurs synchronously with the thrust or during the outward movement of the apex is *systolic*, a thrill asynchronous with the thrust or occurring during the retraction or in-drawing of the apical area is *diastolic*, a thrill that just precedes the thrust is *presystolic*. In case of the apical thrust being impalpable or feeble, the thrill may be timed with the systolic pulsation of the carotid artery in the neck.

Much time should not be spent in timing a thrill. It is easier to time the accompanying murmur. If more than one murmur is present, the louder one is responsible for the thrill.

**SIGNIFICANCE.** The significance of a thrill greatly depends on the (auscultatory) area in which it is felt best. A thrill in the mitral or *apical* area may be either diastolic or systolic. A diastolic thrill in this area is always due to mitral stenosis. A systolic thrill may be felt at times in the apical area in cases of mitral regurgitation or aortic stenosis. A thrill in the apical area is usually best felt in the left lateral decubitus.

A thrill in the *second right space* is usually systolic, and due to aortic stenosis, or aneurysm of the aorta, a diastolic thrill may rarely arise in the aortic area from aortic regurgitation. A thrill in the aortic area is usually best felt with the patient sitting up and leaning forwards.

A thrill in the *second left space* is usually systolic or continuous. A systolic thrill is commoner and may be due to pulmonary stenosis, patent ductus arteriosus or atrial septal defect. A continuous thrill may be due to patent ductus arteriosus or arteriovenous aneurysm. Rarely, a diastolic thrill arises in this area from aortic regurgitation.

A thrill felt in the *third and fourth left interspaces* is usually systolic and due to ventricular septal defect. A thrill in the *tricuspid* area, either systolic or diastolic, may be due to tricuspid regurgitation or stenosis. A tricuspid thrill, unlike all other thrills, is accentuated by deep inspiration.

It is not uncommon particularly in thin nervous individuals to palpate a coarse vibration which may simulate a thrill, but if there is no murmur accompanying the palpatory sensation, it should be considered normal.

A *noncardiac* thrill over the chest or neck may be due to (1) thyrotoxicosis (vascular thrill over the thyroid), (2) vascular malignant tumour, (3) arteriovenous aneurysm or (4) collateral circulation in case of coarctation of the aorta.

**4 Palpable rub or friction fremitus.** In acute or dry pericarditis, the rubbing together of the pericardial layers is usually attended by characteristic friction sounds on auscultation. In some cases, the friction is both audible and palpable, in which case, the sense of vibration imparted to the hand may be termed a palpable rub or friction fremitus. A pericardial rub is usually best felt in the midprecordial area.

**5 Palpable heart sounds.** (a) *Accentuated heart sounds and valve opening sounds* may be felt by the palpating hand. (i) *Mitral tap.* In mitral stenosis the loud and snappy first sound is felt as a tapping apex impulse. (ii) *Palpable opening snap.* The opening snap of the mitral valve in mitral stenosis may be felt as a short clicking impulse just internal to the apex beat. (iii) *Diastolic shock.* In pulmonary hypertension as from mitral stenosis the second sound can be felt in the pulmonary area as a diastolic shock, while in systemic hypertension the loud second sound (Traube's sign) and in syphilitic aortitis the tambour-like second sound may be palpable. (b) *Gallop sounds.* At times gallop sounds may be palpable (and visible) and yet difficult to hear with the

stethoscope (c) *Pericardial knock sound* In constrictive pericarditis a thrust may be felt in early diastole at the time of the third heart sound as rapid ventricular filling comes to an end, and as the systolic thrust is reduced in such cases, it may become the main feature of the cardiac impulse

**6 Carotid artery palpation.** With the patient in the sitting position, the carotid artery on either side is palpated. Besides the character of the arterial pulse, the information obtained in a cardiac case from such palpation is as follows (a) *Presence of a thrill* A systolic thrill is commonly felt in the carotid arteries in aortic stenosis. Also the carotid pulsation in the neck is reduced in such cases (b) *Carotid sinus pressure* This may be of value in the diagnosis of syncope, angina pectoris and the tachycardias. In general carotid sinus pressure should be applied in the recumbent patient and only on one side at a time with the head slightly turned away from the examiner. The pulsations of the common carotid artery should be located and followed to the artery's bifurcation at about the superior border of the larynx. The pressure should be exerted firmly with slight massaging motion for about 5 seconds (i) *Carotid sinus syncope* The carotid artery is compressed towards the spine for 5 to 10 seconds while an assistant counts the pulse and measures the blood pressure. Hypersensitivity of carotid sinus may be one of three varieties. Cardioinhibitory in which there is striking bradycardia, vasodepressor in which there is fall of B P with normal heart rate, cerebral in which there is syncope with normal cardiac rhythm and normal B P (ii) *Angina pectoris* may occasionally be relieved by carotid sinus massage (iii) *Cardiac arrhythmias* In atrial flutter carotid sinus pressure usually slows the ventricular rate to half by producing 4:1 conduction temporarily. In paroxysmal tachycardia a reflex response to carotid sinus pressure may cause a sudden reduction in heart rate. Carotid sinus massage by slowing the heart rate may be helpful in the differentiation of summation gallop rhythm, or the elucidation of diastolic murmurs.

**CAROTID SHUDDER** A coarse vibration at the height of the carotid pulse may be felt in combined A S and A R. It may be associated also with aortic cusp rupture and has also been described in dissection of the ascending aorta.

**7 Tracheal tug (Oliver's sign)** This sign can be elicited by raising the chin of the patient and applying firm upward pressure on the two sides of the cricoid cartilage, with the fingers of one or both hands (Fig 9.12). A downward "tug" felt by the fingers with each beat of the heart suggests the possibility of an aortic aneurysm or mediastinal tumour. It is due to the downward pull exerted by the aneurysmal aortic arch on the left bronchus, later transmitted to the trachea and cricoid.

Pulsation transmitted from the vessels of the neck to the cricoid must not be confounded with tracheal tugging. The movement of the former is forward and backward, of the latter a distinct forward pull with release.

## PERCUSSION

**Historical note** Amongst the notable advances of the eighteenth century may be mentioned the introduction of a new method of clinical investigation, viz. percussion by Joseph Leopold Auenbrugger in 1761. It was the simple expedient of tapping the barrels of wine in his father's cellar to determine their contents that gave Auenbrugger the idea of percussion of the thorax as an aid to diagnosis, a discovery first announced, in 1761, in his *Inventum Novum*. Forty seven years later, Napoleon's physician Corvisart, one of the foremost teachers of medicine in France, "recalled to life" the art of percussion by translating Auenbrugger's long forgotten work of ninety five pages into French.

**Definition** Percussion is a method of examination which depends on the interpretation of sounds heard and the sense of resistance encountered on subjecting the chest to a series of strokes or taps with the fingers or with a special instrument. The sound produced is studied for pitch, intensity and character (auditory percussion), attention being also paid to the sense of resistance encountered by the fingers during the process (palpatory percussion). With good technique and experience, it is possible to advantageously combine a study of these two phenomena, during percussion.

**Methods of percussion** Two methods of percussion are recognized, viz. (1) direct (or immediate) percussion, where the strokes are aimed directly at the chest wall, and (2) indirect (or mediate) percussion (Fig 9 13), where the strokes are aimed at some intermediate object (e.g. a finger) applied to the surface of the chest wall. Although both methods have their uses and advocates, the indirect method of percussion is usually employed routinely, except for percussion over bony structures (like the clavicle) and for special cases where direct percussion is preferred.

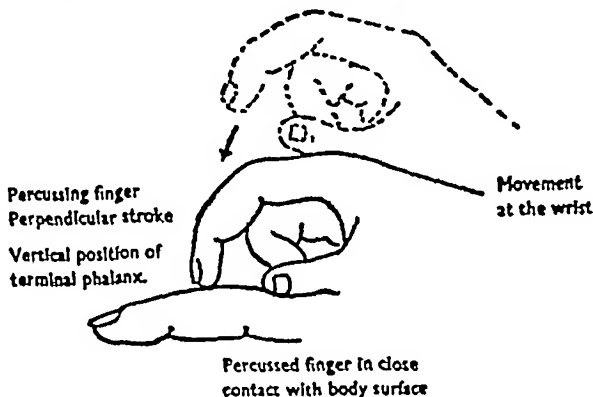


Fig 9 13 Correct technique of indirect percussion

**The "stroke" in percussion** The stroke employed in percussion may be either light or heavy. The use of light or heavy percussion depends partly on the examiner's choice and habit and partly on the region percussed. A heavy stroke is advisable with a thick chest wall, emphysema or deeply embedded heart, whilst a light stroke is preferred when the heart is close to the surface or covered by a thin chest wall. For the front of the chest and the axillary regions, a lighter stroke is required than for percussion of the back. For elderly subjects and children, a lighter stroke is required than for the obese.

or the heavily-built individual. For localized or selective percussion, a light stroke is necessary. The rules of percussion are dealt with in connection with the respiratory system.

**Method of cardiac percussion** (Fig 9 14) Although the major part of the heart is covered by the resonant lung, it is possible by resort to moderately heavy percussion to delineate (somewhat inaccurately) the outline of the heart through the lung tissue. When mapping out the cardiac borders, it is customary to percuss from resonant to 'dull' areas, or centripetally, from surrounding areas towards the heart. This is because the change from a resonant to a dull note is better appreciated by the ear than a change from dull to resonant. Whilst some examiners prefer to hold the pleximeter finger parallel to the cardiac border percussed, others prefer to keep it parallel to the ribs and interspaces (or perpendicular to the cardiac border).

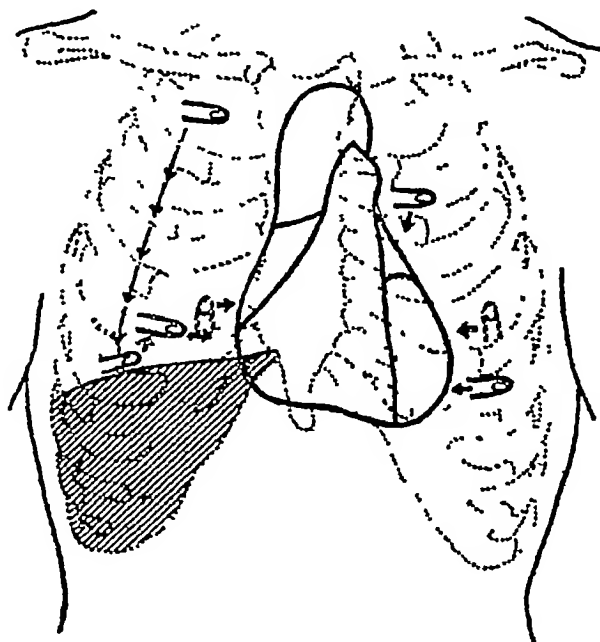


Fig. 9 14 Method of cardiac percussion

**Determination of cardiac borders** (Fig 9 14) The *left cardiac border* is usually the first to be percussed, in the fifth left interspace and from the axilla inwards. A definite impairment or change of note, without actual loss of resonance, appears when the apex of the heart is reached, usually just lateral to the apical impulse. The left border is then similarly percussed in the third and fourth left interspaces. In normal adult males, the cardiac border in the fifth left interspace is usually 7 to 9 cms and in the third space 3 to 3½ cms from the midsternal line. An appreciable displacement of the left cardiac border outwards in the fifth left interspace is suggestive of left-sided enlargement of the heart, pericardial effusion or respiratory disease, such as pleural

effusion on the right side. When the latter measurement (i.e. the distance between the left cardiac border and midsternum in the third left interspace) is appreciably increased, it is suggestive of a prominent left auricular appendage or pulmonary artery as in mitral stenosis, atrial septal defect or patent ductus arteriosus. On the whole, the results obtained with percussion of the left cardiac border are satisfactory (the heart being close to the chest wall), although not accurate.

Regarding the *right border* of the heart, there is doubt as to the reliability of percussion in delineating this border in view of the vibrating qualities of the sternum. However, an enlargement to the right may be recognized by percussion.

The upper margin of liver dullness on the right side must be defined first by percussing from above downwards, in each intercostal space till a change of note is obtained. Percussion is then carried out in an interspace above this level moving inwards towards the sternum, either parallel or at right angles to the right cardiac border. If the percussed border is more than 1 cm. outside the right sternal margin in the third and fourth right interspaces, a diagnosis of cardiac enlargement (of right or left atrium or both), or of displacement of the heart to the right or pericardial effusion should be entertained.

As in the case of the right cardiac border, it is difficult to determine the *upper border* with accuracy. Percussion is started in the first left intercostal space and carried downwards. The percussion note normally changes in the third interspace.

Besides the conventional percussion of the cardiac borders, it is necessary to percuss the second interspace both to the right and left of the sternum to determine dullness due to the presence of an aortic aneurysm, superior mediastinal tumour or of pericardial effusion. Percussion of the third left interspace may reveal dullness due to enlargement of pulmonary artery or of the left atrium or the presence of patent ductus arteriosus.

**Abnormal areas of cardiac dullness.** Abnormalities in extent of the areas of cardiac dullness may be due to (1) physiological factors, such as the phases of respiration (due to diaphragmatic movements), pregnancy or gaseous distension of the abdomen, (2) extrinsic or extracardiac causes, e.g. diseases of the lungs and pleura which displace the heart by pulling or pushing it (such as fibrosis or collapse of lung, pleural effusion or pneumothorax), emphysema (which decreases the area of dullness) or abdominal tumours or ascites, (3) intrinsic or cardiac causes, such as hypertrophy or dilatation of the whole or a part of the heart, ventricular aneurysm or pericardial effusion.

**Area of supracardiac dullness.** This refers to the area of slight dullness, over the upper part of the sternum, caused by the underlying aorta and superior vena cava. The first and second interspaces on either side normally appear resonant. In the event of demonstrable dullness of note over the manubrium sterni or an unmistakable dullness of note in the first or second interspace, the possibility of aortic aneurysm, mediastinal tumour or substernal thyroid should be considered. Because of the sternum, percussion in this area is proverbially difficult and at times unreliable.

**Percussion of the sternum** This has been recommended as useful by some observers. A flat note or dullness underneath the upper part of the sternum is considered suggestive of aortic aneurysm or mediastinal disease, whilst dullness under the lower part is said to indicate a right ventricular enlargement or pericardial effusion.

**Value of cardiac percussion.** Cardiac percussion can still be regarded as a fairly useful method of examination provided its limitations are recognised.

- (a) It is the only method available for gauging the heart size in the absence of the visible and palpable apex impulse and lack of facilities for radiological investigation.
- (b) It is of undoubted value in the diagnosis of a pericardial effusion. Here the percussion note over the cardiac area becomes absolutely dull, the area of dullness increasing in all directions to assume a globular, triangular or pear shape, the dullness extending to the left of the apex beat. The second left interspace is also often dull on percussion.
- (c) In gross left ventricular enlargement, the area of cardiac dullness extends to the left and downwards.
- (d) It may be of some value in the detection of gross enlargement of the right atrium and in detection of displacement of the heart.
- (e) It may help to confirm the presence of an aortic aneurysm.

**Special techniques of percussion** Special methods of percussion have been recommended and used to advantage in cases of heart and lung disease, viz. (1) ortho-percussion, (2) auscultatory percussion, (3) Pottenger's method, and (4) the threshold method. In *orthopercussion*, the left middle finger is flexed to a right angle at the proximal interphalangeal joint before being struck by the plessor finger. In *auscultatory percussion*, the chestpiece of the stethoscope is placed over the sternum just above the xiphisternum and then the skin is lightly scratched from the axillae inwards towards the sternum. The point at which the soft scratching sound becomes suddenly intense corresponds to the cardiac border on that side. In the *threshold method*, the percussion stroke over the area of absolute cardiac dullness is gradually decreased in strength until no sound is produced. Maintaining the same force, percussion is then carried out centrifugally in different directions until the appearance of sound on percussion indicates a transition from dullness to resonance. The *Pottenger's method*, a form of palpatory percussion without a pleximeter, is mainly dependent for results on the tactile sensation conveyed to the fingers. It yields excellent results in the hands of experienced observers.

## AUSCULTATION

**Historical note** A discovery of the greatest importance to cardiology in the early part of the nineteenth century was that of auscultation of the heart and lungs with the aid of the stethoscope by the French physician Laennec. Laennec's discovery represents a rare fusion of intuition, observation and accident. Remembering a well-known acoustic fact, that "if the ear be applied to one end of a plank, it is easy to hear a pin's scratching at the other end", Laennec constructed his primitive monaural stethoscope, a perforated wooden cylinder with a chestpiece at one end and an earpiece at the other. His descriptions of innumerable auscultatory signs, discovered with the aid of his primitive instrument, remain unsurpassed to this day.

Although considered somewhat out-dated or primitive by some, the stethoscope continues to remain and will doubtless remain for many years to come "a source of vital information about the heart".

**Definition** Auscultation is one of the four traditional methods of physical examination and entails listening to the sounds produced within the body in health or disease.



**Methods of auscultation.** As in percussion, there are two methods of auscultation, viz. (1) the direct (or immediate) method, which consists in applying the unded or naked ear to the chest wall, and (2) the indirect (or mediate) method which depends on the use of a stethoscope for listening to the sounds.

**The direct method.** Although the indirect method is employed universally in preference to the direct, the latter method may prove more useful in certain special cases, viz. (1) for listening to faint, high-placed or buzzing sounds (e.g. the faint diastolic murmur of aortic regurgitation or the rattle arising of pneumonia), (2) for bone-conducted sounds, (3) in the absence of a stethoscope, (4) in the case of highly nervous children or babies, and (5) occasionally for timing pulsations of the chest wall with auditory events.

The direct method however has certain disadvantages, viz. (1) it is neither convenient nor accurate with women, (2) it can only be applied to relatively large areas, not being suitable for localized points or valve areas, (3) the suprasternal, infrasternal, and high axillary regions prove inaccessible to it, (4) comparative auscultation of symmetrical areas becomes difficult or faulty, and (5) it does not allow the detection of finer grades of intensity or quality of sounds.

**Type of stethoscope.** The monaural stethoscope represents the earliest stethoscope devised, and the one with which most of the discoveries in auscultation have been made. It is a single wooden tube with a large funnel-shaped earpiece at one end and a small flat earpiece at the other. Although considered obsolete and inconvenient by many, the monaural tube is still favoured in some clinics. Its one great advantage over the binaural stethoscope is that of combining the tactile and aural senses, in such as the timing of auscultatory events (e.g. murmurs, gallop sounds or other extra sounds) with the apex beat.

The binaural stethoscope, which is employed universally, is more convenient to carry, easy to apply over local or inaccessible regions of the chest, and has a wide range of audibility.

**Type of earpiece.** Many different types of earpiece have been devised with actual or alleged advantages, the most popular types being (1) the bell-type with a funnel or bell-shaped earpiece of metal or hard rubber (the latter being better to avoid by patients in cold weather), (2) the diaphragm type, with a shallow cup and a diaphragm, and (3) the combined type of earpiece, where either the bell type or diaphragm type of receiver can be put into operation, at will, by means of a lever.

The bell-type of earpiece is particularly useful for (1) detecting low-placed murmurs and sounds (e.g. the mural diastolic murmur or the third heart sound) and (2) auscultating local areas (e.g. the suprasternal region) or interspaces (especially in children with narrow interspace).

The diaphragm (Bowles) type is useful for (1) faint or high-placed sounds (e.g. the diastolic murmur of aortic regurgitation or early pneumonic sounds) and (2) is more convenient for auscultating the back and axillary regions.

The combined earpiece is even more useful, combining as it does the advantages of both the bell and the diaphragm types of earpiece.

**Efficiency in auscultation.** This depends on several factors. (1) The first is the stethoscope. Care should be taken to see that the earpieces fit snugly into the ears, are applied at the right angle, and are of correct size. The chest-piece must be properly applied and warm (so as not to promote shivering and adventitious sounds); the rubber tubes must not be blocked or cracked, and the joints not loose or broken, the diaphragm must be intact and the springs not too tight. The selection of a good stethoscope demands care and attention to the above-mentioned points, this will more than repay the extra time and

care involved. (2) The other factor is “the man behind the stethoscope” The most important requirements for efficiency in auscultation are the experience and knowledge of the examiner, his ability to discard or disregard unimportant sounds (such as extraneous noises, muscle, hair and chest wall sounds) and his selective powers of concentration or attention. Sufficient time must be reserved for each area of auscultation and separate attention reserved for each phase of the cardiac cycle. The patient must be completely comfortable and in an ideal position for auscultation.

**Auscultatory areas** It is traditional to recognize four main auscultatory or valve areas over the front wall of the chest. These areas do not correspond to the anatomical situations of the underlying valves (Fig 9 15), but represent points at which sounds or murmurs originating in these valves are heard best. The four classical valve areas are (1) *mitral* or *apical* area, or region of the cardiac apex, normally situated in the fifth left interspace in or near the mid-clavicular line, (2) *aortic area*, at the inner end of the second right interspace or costal cartilage, (3) *pulmonary area*, at the inner end of the second left interspace, and (4) *tricuspid area*, at the lower end of the sternum near the

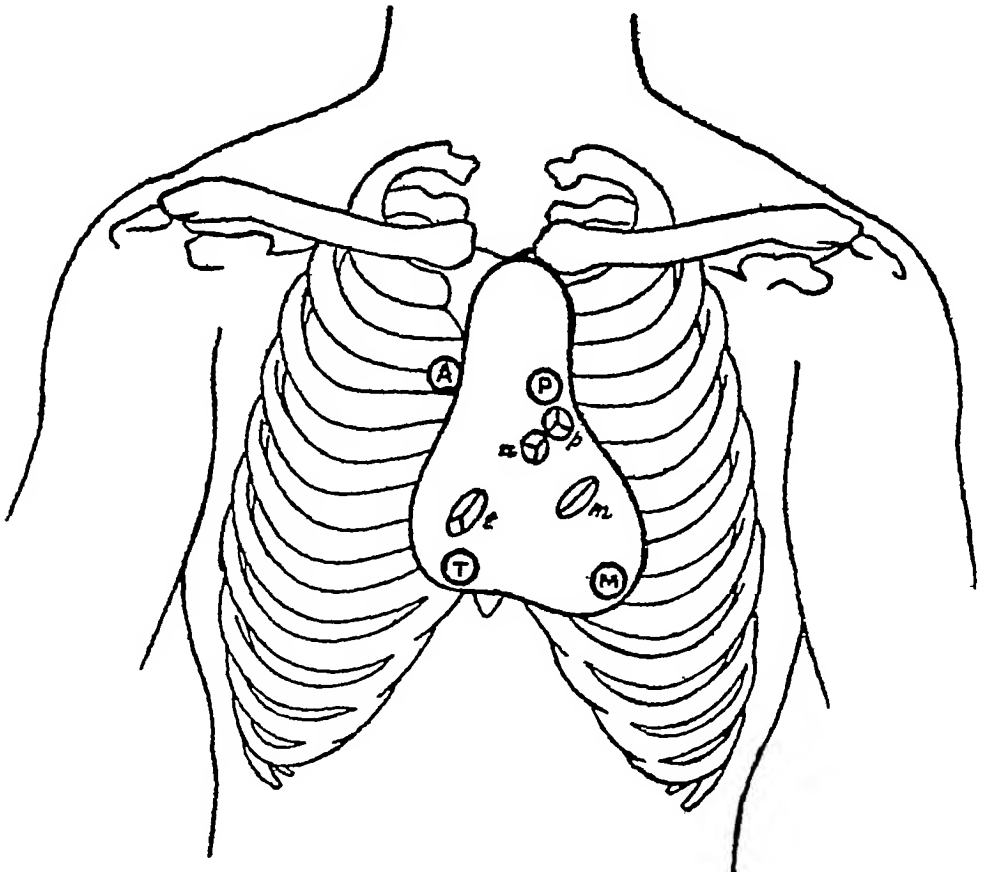


Fig 9 15 Auscultatory areas and anatomical sites of heart valves

ensiform cartilage, or along the left border of the sternum at the level of the fourth intercostal space

Nomenclature of the traditional areas of auscultation, as it exists today, suffers from certain inadequacies. Whilst the terms "aortic area" and "pulmonary area" are suitable, in that they do represent acoustic events involving the aorta and aortic valve on the one hand, and the pulmonary artery and pulmonary valve on the other, the same is not true for the "mitral" and "tricuspid" areas, where important auscultatory phenomena, independent of the mitral and tricuspid valves, are frequently encountered. Under the circumstances, the alternative designations of "apical area" (corresponding as it does to the clinical apex of the heart) and "low sternal area" are recommended in place of the classical "mitral" and "tricuspid" areas respectively.

Restriction of auscultation of heart sounds and murmurs to the four traditional areas leaves examination of the cardiovascular system incomplete. In order to facilitate clinical diagnosis, the following additional auscultatory areas or sites are recommended for routine employment: (1) *Mid-sternal area* (*second aortic area* or *Erb's area*), which corresponds to the inner end of the third left interspace, and where the soft early diastolic murmur of aortic regurgitation and the pansystolic murmur of ventricular septal defect are frequently best heard. (2) *Area just below the inner end of the left clavicle*, a site of election for the characteristic continuous murmur of patent ductus arteriosus. (3) *Cervical area* or *area over the neck vessels* (carotid arteries) on either side. The systolic murmurs of aortic valve disease, dilated aorta, occlusive arterial or cerebrovascular disease and hyperdynamic circulatory states are frequently well heard here. (4) *Interscapular area*, between the two scapulae and along or to the side of vertebral column. Murmurs of aortic coarctation and aneurysms of the descending thoracic aorta are often heard best in this area. (5) *Infrascapular areas*, below the scapulae on the two sides and over the lung bases, are the sites of election for various murmurs, indicative of collateral circulation of coarctation of the aorta and of pulmonary fistula, truncus arteriosus, etc. (6) *Cranial areas*. The skull and orbits, on auscultation, may afford diagnostic auscultatory evidence of cerebral arteriovenous fistulae, angiomas or occlusive vascular disease. (7) *Abdominal areas*. Auscultation over the epigastrium, renal areas and flanks, may bring to light systolic or continuous murmurs indicative of renal artery stenosis with hypertension and the venous hums of hepatic cirrhosis or venous obstruction. (8) *Peripheral arterial sites*. Routine auscultation over certain major arteries of the body, such as the femorals, popliteals and subclavians, may disclose murmurs of great diagnostic significance.

In view of the fact that the pathognomonic presystolic rumbling murmur of mitral stenosis is frequently localized to a minute area just internal to the classical mitral area, this area must always be auscultated with care.

For special cases, where numerous adjacent areas of the chest wall require auscultatory probing, the method of "inching", or inch by inch auscultation along lines connecting valve areas, may be employed to advantage

*Special postures, and effect of exercise and respiration, and other manoeuvres* These may prove necessary from time to time, during cardiovascular auscultation. *Posture and exercise* Initially auscultation should be carried out with the patient supine during quiet respiration, then asking the patient to take a long slow breath and stopping and finally breathing out and holding the breath in full expiration. The murmur of mitral stenosis may be best heard or only heard with the patient lying in the left lateral position especially if the blood flow is increased by exercise. The murmur of aortic regurgitation is best heard with the patient sitting up or leaning forward and with the breath held in expiration. It is sometimes only heard with the naked ear applied to the chest. *Respiration* The effect of respiration is of particular importance in regard to the two components of the second sound and the tricuspid murmur. Right sided cardiac events are usually louder during inspiration and left sided events during expiration. *Other methods or manoeuvres* necessary include use of diaphragm or bell type of chest piece, Valsalva manoeuvre, carotid sinus pressure, isometric hand grip, and use of pharmacological agents such as amyl nitrite and vasopressor drugs

## HEART SOUNDS

It is customary today to recognize and describe several varieties of "heart sounds" on the basis of clinical auscultation and phonocardiography or graphic registration. They have been classified, according to the mechanism of their production, as follows

### 1 *Valve closure sounds* (A-V and semilunar valves)

First heart sound	{ Mitral component Tricuspid component
Second heart sound	{ Aortic component Pulmonary component

### 2 *Valve opening sounds* (A-V valves)

- Opening snap of mitral valve
- Opening sound of tricuspid valve

### 3 *Ventricular filling sounds*

3rd heart sound	{ Right ventricular Left ventricular
Atrial (4th) heart sound	{ Right atrial Left atrial
Summation sound	

4 *Ejection sounds*

Early systolic ejection sounds  $\left\{ \begin{array}{l} \text{Aortic} \\ \text{Pulmonary} \end{array} \right.$

Post-ejection sounds (clicks)

5 *Other sounds*

Pericardial knock sound

Artificial valve sounds

Pacing sounds

A detailed study of the various heart sounds, in health and disease, is imperative for a proper understanding and diagnosis of ailments of the heart. It is important to realize that (a) normal heart sounds do not rule out the possibility of a diseased heart, (b) physiological variations, such as the splitting of a normal heart sound or the addition of an extra sound such as the third heart sound, may be encountered in a perfectly normal heart.

Normal heart sounds On clinical examination, in normal subjects, the only heart sounds audible are the first and second heart sounds in all subjects, and the physiological third sound in a fair percentage of young children and adults. These sounds are referred to as normal heart sounds. A fourth heart sound can be recorded phonocardiographically but is rarely audible (except at times in cases of complete heart block).

FIRST HEART SOUND represents the onset of cardiac systole, is synchronous with the cardiac impulse and is best heard at the apex.

SECOND HEART SOUND is best heard at the base. Normally, although the aortic component of the second sound (indicative of closure of aortic valve) slightly precedes the pulmonary component, the two elements of the sound appear fused, at least during expiration and give rise to auditory impression of a single sound. The aortic component although audible all over, is best heard over the aortic area and apex, the pulmonary component is usually heard over a small area, in the second and third left interspaces.

Mechanism of production of heart sounds It is customary to regard the production of heart sounds as due to vibrations resulting from sudden alterations in the velocity of the blood stream—valve closure suddenly stops or reverses the movement of blood and is the most important factor in the genesis of normal heart sounds.

First heart sound The first heart sound is almost entirely due to closure of the mitral and tricuspid valves in that order when the pressure in the ventricle rises above that in the atrium. Normally the mitral and tricuspid components of the first sound appear fused and give the auditory impression of a single sound, although the mitral component precedes the tricuspid component by a fraction of a second (0.02-0.03 sec). Splitting of the first sound may be encountered in healthy children and young adults and is best heard

over the tricuspid area This has been phonetically represented by the syllable "turupp"

If the A-V valves are prevented from closing, the first sound does not completely disappear This is likely to be due to vibrations set up in the ventricular walls, like the noise produced when a piece of string is pulled out suddenly Atrial contraction also contributes, since as the A-V conduction time is diminished, the sound becomes louder A haemic factor may be an additional contributory element

*Second heart sound* This results from sudden closure of the aortic and pulmonary valves when ventricular pressure falls below that in the great vessels The aortic component of the sound slightly precedes the pulmonary component Splitting of the components is fairly commonly heard in normal healthy children and young adults It is usually best heard and split wider during inspiration (Fig 9 17), an act which by causing increased filling prolongs right ventricular systole and delays the appearance of the pulmonary component There is also some evidence that left ventricular ejection is shortened during inspiration possibly due to inspiration holding back venous blood in the lungs The cardiac rhythm produced by a splitting of the second sound has been compared to the hoof-beats of a "cantering horse", and termed canter rhythm or "bruit de rapple"

For clinical purposes the aortic component can be identified in the aortic and mitral areas and the pulmonary component in the pulmonary area Normally in adults  $A_2$  is louder than  $P_2$  because diastolic pressure in the aortic exceeds that in the pulmonary artery In children  $P_2$  may be louder or as loud because the pressure difference is not so much and the pulmonary artery is relatively large and closer to the chest wall Evidence has been put forth to suggest that like the first sound, the second sound occurs shortly after valve closure

*Natural rhythm of the heart* (Fig 9 16) For practical purposes it is customary to regard the natural rhythm of the heart as a "dual rhythm" and describe

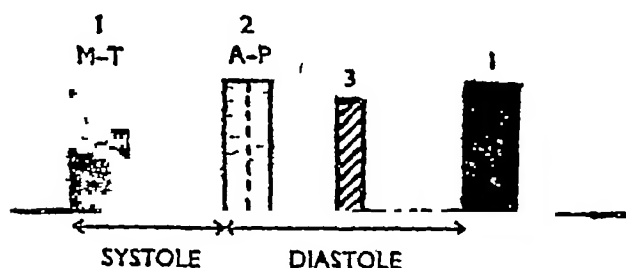


Fig 9 16 Normal cardiac rhythm The third sound may or may not be heard

only two heart sounds, viz. the first and second heart sounds The normal cardiac rhythm corresponds to "triple time" in music with two beats followed by a silent beat or pause In the mitral and tricuspid areas the accent is on the first beat, whilst in the basal areas

it is on the second, the natural rhythm of the heart is usually phonetically described as "lub-dub", "lub-dub", or as "lubb-tup", "lubb-tup"

**COMPARATIVE STUDY** Compared to the first sound, the second sound is (1) shorter in duration (the duration of the normal first and second sounds being approximately 0.14 and 0.11 seconds respectively), (2) higher in pitch, and (3) snappier or more sudden in character.

There are two reasons why the second sound is higher in pitch and has a higher frequency than the first sound. (1) The elasticity of the semilunar valves and arterial trunks is much lower than that of the auriculoventricular valves and ventricular walls, in any mechanical vibrating system, the lower the elasticity the higher the rate of oscillation. (2) The volume of the blood being much less in the arteries than in the ventricles, the inertia of the vibrating mass is much less in the former with a correspondingly higher frequency of vibration.

The second sound is shorter in duration than the first because the higher frequency of oscillation in the former causes an earlier and more rapid damping out of the sound by the viscosity of the vessel and heart walls.

The second sound displays a more *snappy*, or valvular quality than the first.

**PAUSES OR TIME INTERVAL.** During each normal cardiac cycle, there are two pauses or time intervals between the heart sounds. (1) a short systolic pause, between the first and second sounds, and (2) a long diastolic pause between the second and first heart sounds. In rapidly beating hearts, the diastolic pause is usually shortened to a greater extent than the systolic pause.

**IDENTIFICATION OF HEART SOUNDS** A distinction between the first and second heart sounds permits a proper evaluation of each sound and facilitates the timing and recognition of heart murmurs. The first heart sound is usually identified by (1) its character or quality, (2) lower pitch, (3) longer duration, (4) greater intensity over the apex, and (5) the pause preceding the sound being longer than the one following it.

The first sound by auscultation may be difficult or impossible to identify in case the heart rate is unduly rapid or the observer inexperienced. In such cases, it may be recognized by observing or palpating the apex impulse or pulsation of the carotid artery in the neck. Although a short time-lag exists between the first sound and the carotid pulsation (of 0.09 second), for practical purposes, this may be ignored. The radial pulse must never be employed for identification or timing of cardiac events, since appreciable time is lost in the transmission of the pulse wave to the periphery.

**RELATIVE INTENSITY OF NORMAL SOUNDS** The relative loudness or intensity of the first and second heart sounds depends to a great extent on the part of the chest auscultated. Over the mitral and tricuspid areas, the first sound is relatively louder than over the basal areas. The second sound is usually heard best over the pulmonary area in children and young adults, and over the aortic area in the middle-aged and elderly.

**EFFECTS OF NON-CARDIAC FACTORS ON HEART SOUNDS** A *proportionate accentuation* or increase in loudness of the heart sounds may be due to a thin chest wall, excitement, emotion, fever, or exercise which results in an increase

of blood flow, young age or retraction of one or both lungs by disease, with uncovering of part of the heart

The heart sounds may display a peculiar *metallic character* in case of pneumothorax or gaseous distension of the stomach or intestine

A *proportionate attenuation* or diminution of intensity of sounds resulting in faint, muffled or impure sounds may be due to a thick chest wall, emphysematous lung or pericardial effusion

Change of posture, such as stooping or bending forwards or sideways or lying sideways in bed or Valsalva manoeuvre, may bring about an alteration in intensity of the heart sounds

Severe thoracic deformities, such as scoliosis, kyphoscoliosis and funnel chest, may bring about a proportionate accentuation or attenuation of sounds depending on the proximity or otherwise of the heart to the region of the chest auscultated

GRAPHIC METHOD OR RECORDING HEART SOUNDS (Fig 9 17) The heart sounds are represented by rectangular blocks, placed vertically, the height representing loudness or intensity of sound, the width corresponding to its

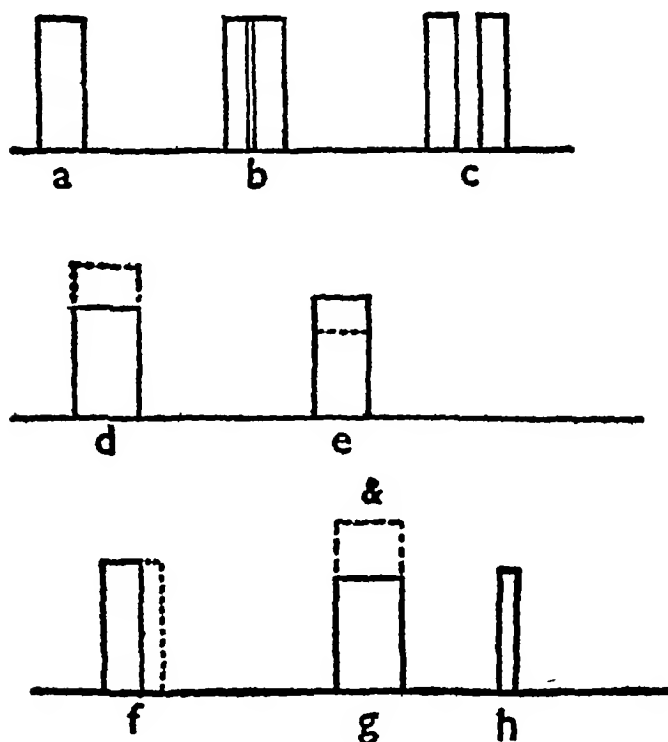


Fig 9 17 Graphic recording of heart sound abnormalities (a) Normal sound (b) Narrow splitting (c) Wide splitting. (d) Accentuated sound. (e) Attenuated sound (f) Increased duration or prolongation (g) Accentuation with tympany or metallic note (h) Extra sound

duration Each diagram is made up of the first and second sounds of one cardiac cycle, followed by the first sound of the next cycle, thus indicating the two phases of systole and diastole Narrow and wide splitting of heart sounds are shown as in Fig 9 17 Extra sounds are shown by rectangular blocks

Third heart sound In most people two sounds are heard with each cardiac cycle However, in a fair number of normal healthy children and in a proportion of healthy young adults, there is a



superadded third sound.

The *physiological third heart sound*, first described by Gibson in 1907, is usually attributed to the sudden distension of the left ventricle or vibration of its walls by the sudden inrush of blood during diastole, with or without a sudden tautening of the mitral valve leaflets and chordae tendinae. It occurs approximately 0.15 second after the second sound and corresponds to the end of the phase of rapid ventricular filling. Commonly encountered in children (70 to 80 per cent) and young adults (30 to 50 per cent), the physiological third sound is seldom if ever encountered after the age of 40 years, in the middle-aged and elderly, its presence is usually indicative of heart disease. It is common in pregnancy.

The normal third heart sound is usually (1) best heard at the apex or just internal to it, (2) localized to a small area, (3) best heard or only heard during recumbency (being frequently inaudible in the upright position), (4) soft, short and low of pitch, (5) frequently accompanied by a palpable shock (perceptible near the apex), (6) varies with respiration, being best heard at the onset of expiration, and disappearing during held inspiration, (7) increased by exercise, (8) best heard with the bell-type of chestpiece, (9) accentuated by raising the legs or by pressure on the abdomen (measures which increase venous return) and (10) best heard in the left lateral position. It may not be present with every cardiac contraction. (11) It is never split.

The third heart sound is responsible for an innocent or harmless variety of triple rhythm (phonetically represented by "lub-dub-da")

**MECHANISM OF PRODUCTION** The production of third sound is attributed to the sudden distension of the left ventricle or vibrations of its walls by the sudden inrush of blood during diastole with or without abrupt tautening of the mitral valve leaflets and chordae tendinae.

**Fourth heart sound.** It occurs late in diastole (presystole) and is recorded phonocardiographically but an audible fourth sound is abnormal. If the A-V conduction time is long enough, the atrial sound will be heard distinct from the following first sound.

**Pathological variations of heart sounds** In disease the heart sounds may be modified or altered in one of several ways. They may become (1) loud or accentuated, (2) soft, diminished or attenuated, (3) absent or masked, (4) split or (5) delayed, or (6) variable in intensity, or (7) single.

**First heart sound.** The *intensity* of the first sound depends on (1) the position of the atrioventricular valve leaflets at the onset of ventricular systole, the more widely open the valve at the onset of the systole (from continuation of blood flow from atrium to ventricle) the greater the force of closure and the louder the sound produced. (2) *Mobility* of the cusps, gross fibrosis or calcification of the mitral valve is associated with soft first sound. (3) *Adequate*

A-V valve cusps Adequacy of valves halts regurgitant flow (4) Rate of ventricular contraction, slow contraction of a partly functioning left ventricle may cause a soft first sound All these mechanisms help to explain the varying intensity in various clinical conditions

### Abnormalities of the first heart sound

ACCENTUATION OF THE FIRST SOUND, as a rule most marked at the apex, may be due to

A *Extrinsic causes* such as close approximation of the chest wall as in cases of thin chest wall and spinal deformity

#### B *Intrinsic causes*

(1) Mitral stenosis The first sound is typically loud, sharp and high-pitched, described as “snappy” or “slapping” unless the mitral valve is grossly fibrosed, calcified or regurgitant The loudness is due mainly to the high-pressure gradient between the left atrium and ventricle and the slow filling of the ventricle keeping the mitral cusps wide open until the very end of the diastole thus resulting in a forcible valve closure during systole The thickening of the valve ring and of the anterior valve leaflets also contributes to the loudness and high pitch of the sound The loud first sound is due to a sudden and forcible excursion of the bellies of the mitral valve leaflets towards the left atrium, during the phase of isometric ventricular contraction A loud, sudden and short first sound is often the earliest or presenting sign of mitral stenosis, and as such is of great diagnostic value The intensity of the sound, although mainly dependant on the large anterior leaflet of the valve, is not proportional to the severity of stenosis

With advancing mitral regurgitation in case of stenosis, the first heart sound tends to become softer or attenuated because of (a) diastasis or equalization of left atrial and left ventricular pressures, resulting in reduced (forward flow) pressure on the mitral valve leaflets, (b) disease or destruction of valve leaflets, frequently associated with the regurgitant condition and (c) partial or complete “enveloping” of the first sound by the long pansystolic murmur of regurgitation In case of atrial fibrillation, the loud first sound of mitral stenosis (particularly of severe stenosis) persists as a rule, despite absence of left atrial systole This is due mainly to persistence of a high end-diastolic pressure gradient between the left atrium and ventricle (because of the stenotic condition), the forward flow from the left atrium exerting pressure against the valve cusps The accentuated first sound of mitral stenosis usually persists after a mitral commissurotomy operation unless atrial fibrillation, myocardial failure, iatrogenic mitral regurgitation or partial heart block happens to reduce its intensity

The accentuated first sound of mitral stenosis is classically associated with a loud and split second sound in the pulmonary area, an opening snap and a localized diastolic murmur near the cardiac apex

(2) *Shortened atrioventricular conduction time* When the P-R interval of the electrocardiogram is shortened, the atrial contraction forces the valve cusps widely open by a great surge of blood just prior to ventricular systole, resulting in a loud and sudden first sound. With a P-R of 0.16 second, the first and second sounds are of equal intensity, whilst with P-R interval of 0.14 second or less, there is an increase in the relative intensity of the first sound. The loudest first sound can be heard when P-R interval is 0.1 to 0.12 seconds. The importance of the relationship of the first sound to the P-R interval is particularly apparent under the following circumstances: (a) Early detection of myocardial involvement or carditis in suspected cases of rheumatic fever. (b) In the follow-up of cases of rheumatic carditis, replacement of a feeble first sound by a loud or normal one being indicative of clinical improvement and vice versa. (c) In case of repeated attacks of syncope or dizziness, attention may be directed to the possibility of Adams-Stokes syndrome as the cause of attacks, by a weak first sound indicative of first degree A-V heart block. (d) When myocardial damage is wrongly diagnosed on the basis of a weak first sound. Correct diagnosis of a prolonged P-R interval as the cause of the phenomenon, may save the patient from the stigma of serious heart disease and unwarranted invalidism.

A weak first sound, in conjunction with a normal heart and an atrial sound in presystole, should suggest the possibility of first degree heart block with increased P-R duration.

(3) *Cardiac dysrhythmias* (a) *Tachycardia* Shortening of diastole from tachycardia keeps the valve open till the end of diastole and the first sound is accentuated. (b) In the Wolff-Parkinson-White syndrome, or the so-called "accelerated conduction", the loudness of the first sound depends on the shortening of the P-R interval. In this condition, the two ventricles are excited asynchronously, the right ventricle before time and the left ventricle at its usual time. Since the mitral or louder component of the sound is unchanged, the first sound may remain more or less unaffected despite the presence of incomplete right bundle branch block. In the L G L or Lown-Ganong-Levine syndrome of short P-R interval with normal QRS complexes, usually observed in women over 30, accentuation of the first sound is almost invariably present. (c) In atrial flutter and paroxysmal tachycardia, the first sound is frequently accentuated.

(4) *States of hyperkinetic circulation* Shortening of diastole from increased flow in high output states such as thyrotoxicosis also leaves the valve open till the end of diastole.

(5) *Left to right shunt* with increased A-V blood flow and prolonged ventricular filling.

(6) *Myxoma of left atrium* is a rare cause. It is likely that the tumour prolapsing in diastole through the valve orifice together with the raised left atrial pressure causes delay in valve closure.

(7) *Floppy valve syndrome* Here a likely factor for increased intensity is the large area of the redundant valve (This can be compared to loud tricuspid closure sound of Ebstein's anomaly)

**DIMINISHED INTENSITY** Soft or attenuated first sound may be due to

A *Extrinsic causes* such as thick chest wall, emphysema or pericardial effusion leading to a further separation of the heart from the chest wall

B *Intrinsic causes*

(1) *Bradycardia* Prolongation of diastole when the heart rate is slow allows time for the leaflets to approximate together

(2) *Increased atrioventricular conduction time* An increased duration of the P-R interval, of over 0.16 second, leads to a relative diminution in the intensity of the first sound. The valve cusps, being partially approximated and taut, close gently or more silently at the onset of systole because of prolonged A-V conduction time. In acute rheumatic fever and rheumatic pericarditis, the attenuation of the first sound is due to prolongation of the P-R interval

(3) *Mitral stenosis with grossly fibrosed or calcified valve leaflets*

(4) *Mitral regurgitation* With severe mitral regurgitation the mitral cusps cannot hold back the regurgitant flow and since there is sudden halt of these cusps with ventricular systole, the first sound is soft

(5) *Aortic stenosis* The first sound is usually soft perhaps because atrial contraction is vigorous and the ventricle stiff so that presystolic closing factors are greater than normal

(6) *Left bundle branch block* Here the slow rise of the left ventricular pressure pulse is partly responsible

**VARIABLE INTENSITY** This mainly results from dissociation between atrial and ventricular contraction

(1) *Complete heart block* Here variation of the intensity of first sound is a better sign than the cannon waves of the venous pulse. Varying intensity with regular rhythm indicates varying A-V relationship, and different rates of rise of pressure in the left ventricle. It should be mentioned that cannon sounds are not synchronous with cannon waves

(2) *Atrial fibrillation* Because of varying rhythm and changing duration of diastole, and varying position of A-V valves the first sound varies in intensity

(3) *Atrial flutter with changing block* With fixed degree of block there is no change in the intensity of the sounds, but with varying degree of block such as induced by carotid sinus stimulation, variation in intensity of the first sound occurs due to variation in the f-R interval just preceding the ventricular contraction

(4) *Ventricular tachycardia* The first sound varies in intensity, being louder or softer because of the slower contraction of the atria in comparison to the ventricles, with resultant change in the position of the A-V valves at moment of ventricular systole. A changing intensity of the first sound, associated with lack of slowing during carotid sinus stimulation, in case of paroxysmal tachycardia, is highly suggestive of ventricular tachycardia.

(5) *Ectopic beats* Ventricular extrasystoles are associated with a short diastole and usually a loud first sound. However, if there is a very weak ventricular contraction the A-V valves do not close forcibly and the first sound tends to be soft or may be absent. In atrial extrasystole the first sound may be louder or softer than normal.

(6) *Wenckebach block* The gradual lengthening of the P-R interval in successive beats in this arrhythmia causes successive diminution in the intensity of the first sound until the beat is dropped.

(7) *Myocardial infarction* or myocarditis

(8) *Cardiomyopathy*

(9) *Cardiac alternans* Alteration in intensity of heart sounds may be present with pulsus alternans.

(10) *Shock or peripheral failure* In moribund patients, during myocardial infarction or myocarditis, after prolonged fevers and in myxoedema, the first sound may be diminished in intensity because of a slower and less abrupt closure of the valve cusps.

**MASKING** Masking means inability to hear a sound well because of interference by another sound. The first sound may appear absent, when masked or accompanied by a loud systolic murmur. Careful auscultation of the heart usually reveals the true state of affairs in such cases.

**SPLITTING** A heart sound is split when its component parts or events occur asynchronously or are separate. Splitting of the first heart sound may be due to asynchronous closure of the mitral and tricuspid valves, or to asynchronicity of the components or events causing the sound.

*Physiological splitting* of the first sound may be heard, at times, in perfectly normal individuals. The splitting is best heard near the lower end of the sternum because this site is closest to the relatively softer tricuspid component of the first sound. It is also best heard with the breath held in full expiration. Physiological splitting of the first sound must be distinguished from an atrial sound, aortic or pulmonary ejection sounds or clicks, right bundle branch blocks and the presystolic murmur of mitral stenosis.

*Pathological splitting* of the first sound may be due to (a) *Electrical delay* causing abnormal ventricular asynchrony, the most common cause arising in the left ventricle. (1) In complete right bundle branch block, a wide splitting of the first sound, is due to an abnormal delay of the tricuspid component of

the sound as the result of a delay in onset of the rise of pressure in the right ventricle (2) Ventricular extrasystole arising from left ventricle (3) Idio-ventricular rhythm of RBBB pattern (4) Artificial pacing from L V electrode enabling a known site of stimulation to be varied in the same patient (5) Lone atrial fibrillation (6) Patients subject to paroxysmal tachycardia due to pre-excitation of the ventricles

(b) *Mechanical delay* (1) Mitral stenosis (2) Myxoma of left atrium (3) Ebstein's anomaly (delayed or loud tricuspid closure sound).

*Delayed first sound* The first heart sound at the apex may be somewhat delayed in cases of mitral stenosis, occurring a fraction of a second after the cardiac impulse. This is due to the fact that in mitral stenosis, the early part of systole is at times utilized in bringing up the left ventricular pressure to that of the left atrium, resulting in a delay of the mitral valve closure and mitral component. The mitral component may actually follow instead of preceding the tricuspid component thus resulting in the so-called delayed first sound of mitral stenosis.

**TIC-TAC RHYTHM** (Tic-toc rhythm) Shortening of the diastolic pause due to rapid heart rate and alteration of the first heart sound, so that it is short and sharp and resembles the second heart sound, gives rise to a rhythm resembling the ticking of a clock. Since this is comparable to the sounds of the foetal heart, it is also called embryocardia or foetal rhythm. Tic-tac rhythm commonly occurs in states of shock, it may be heard in myocarditis.

**Second heart sound** The second heart sound is due to the sudden closure of the aortic and pulmonary valves. It is a composite sound, made up of the aortic and pulmonary components. The two components are normally fused during expiration and give rise to the auditory impression of a single sound. During inspiration, because of increased filling of the right ventricle, systolic contraction of that ventricle is somewhat prolonged (increased stroke volume), and results in a delayed closure of the pulmonary valves, because of the asynchronous closure of the aortic and pulmonary valves, there results, normally, an inspiratory splitting (normal splitting) of the second sound (Fig 9 18)

The second sound is usually best heard at the base, where it is normally louder than the first sound. In children and young adults (up to the age of 30 or 40 years), the second heart sound is louder in the pulmonary area than in the aortic area. The reverse is true in the middle-aged and elderly. The increasing relative intensity of the second sound in the aortic area with age is attributable to (1) increasing intra-aortic or arterial pressure, (2) sclerotic changes within the walls of the aorta and aortic valves, and (3) alterations in the relative positions of the aorta and pulmonary arteries.

Of the two components of the second sound, the aortic arises just a fraction of a second earlier than the pulmonary, is audible all over the precordium although best heard over the aortic area and apex, and is louder or more intense than the pulmonary component. The latter is usually heard over a small

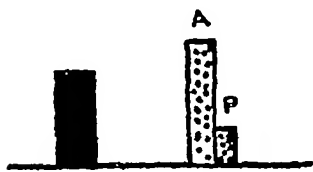
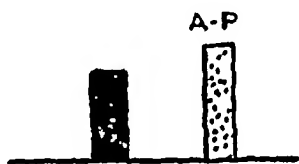
Normal  
Narrow splitting  
during inspiration.



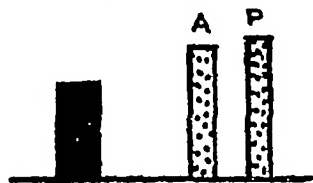
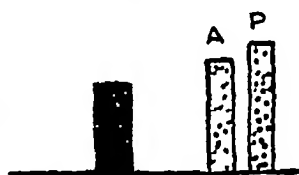
Fallot's tetralogy  
Pulmonary component  
absent. Absence of  
normal splitting



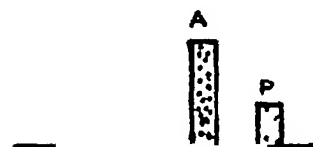
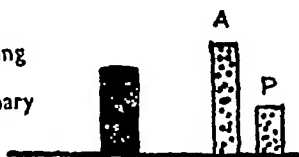
Hypertension  
Aortic component  
masks pulmonary  
Absence of normal  
splitting



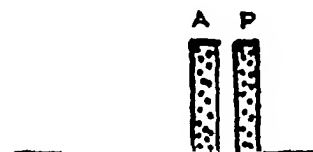
Right bundle branch  
block Wide splitting  
during inspiration



Isolated pulmonary  
stenosis Wide splitting  
during inspiration  
with a feeble pulmonary  
component



Atrial septal defect  
Fixed splitting



Left bundle branch  
block or Aortic stenosis  
Paradoxical splitting  
Splitting during  
expiration



Pulmonary hypertension  
Abnormally narrow or  
normal splitting with  
loud pulmonary component  
and ejection click.



Fig 9 18 Physiological and pathological splitting of the second sound during respiration

area of the chest and best heard over the pulmonary area. The wider area of distribution of the aortic component is due partly to its being louder (because of the "closing pressure" being higher in the aortic than the pulmonary artery) and partly to the aorta being located deeper within the chest than the pulmonary artery

*Normal splitting* Normally, the second heart sound is a single or composite sound during expiration and a double sound or "split" sound at the peak of inspiration or soon after. The inspiratory splitting of the sound is best heard in children and young adults, in the second and third left interspaces and during ordinary breathing. In normal children and young adults expiratory splitting may sometimes be heard in the recumbent posture but nearly always disappears on sitting or standing up. However if splitting can be appreciated when sitting or standing it suggests presence of heart disease. In adults splitting which can be heard on expiration is always abnormal.

*Absence of respiratory variation in splitting* This may be due to (1) R V disease or failure abolishing its ability to increase its stroke volume and thus the physiological inspiratory delay of  $P_2$  may disappear (However R V failure is usually associated with prolonged R V systole and delay of  $P_2$  with resultant wider splitting) (2) Atrial septal defect. The widely separated  $A_2$  and  $P_2$  delay equally on inspiration causing a fixed split (3) Ventricular septal defect if very large causes equal delay of  $A_2$  and  $P_2$

*Identification of the aortic and pulmonary components of the second sound* This helps to (a) identify nearby sounds and murmurs, (b) compare the duration of R V and L V systole in the same cycle, (c) estimate the effect of respiration on the loading of the two ventricles, and (d) compare the intensity of the two components to help in the diagnosis of pulmonary hypertension

*Abnormalities of second sound* The intensity or loudness of the second sound depends on (1) the force of closure of the aortic valve, which determines its aortic component, and the force of closure of the pulmonary valve, which determines its pulmonary component, (2) the proximity of the aorta and pulmonary artery to the chest wall, being closer to the surface when the vessels are dilated or enlarged, (3) the thickness of the intervening lung tissue or chest wall interfering with the transmission of the sound, and (4) masking. A loud murmur such as that of aortic or pulmonary stenosis or patent ductus arteriosus may mask a normal or attenuated second sound.

**ACCENTUATION** The *aortic component* of the second sound may be accentuated in (1) Systemic hypertension from any cause, this factor acts by increasing the force of closure of the aortic valve (2) Aortic dilatation from syphilitic aortitis or atheroma, the dilated vessel being closer to the chest wall. The loud second sound may display a characteristic musical quality ("tambour" sound) in such cases as well as on occasional cases of diastolic hypertension (3) Physical exercise or emotional excitement by raising the pressure



within the aorta (4) Some cases of aortic regurgitation The sudden fall of pressure towards the end of systole in such cases may bring about an abrupt closure of the aortic valve with a high-pitched, loud aortic sound (5) Valvular disease of rheumatic or atherosclerotic origin In such cases, when the aortic valve, though thickened, remains flexible and mobile, the second sound tends to become accentuated

The pulmonary component may be accentuated due either to conditions associated with hypertension of the pulmonary circulation or to dilatation of the pulmonary artery The main causes of such accentuation are (1) mitral valve disease, (2) primary pulmonary hypertension, (3) pulmonary hypertension secondary to atrial septal defect, ventricular septal defect, patent ductus arteriosus, A-V canal, aortic septal defect or recurrent pulmonary emboli, (4) left ventricular failure, as in hypertension or coronary artery disease, (5) chronic lung disease, such as emphysema and massive lung fibrosis, and (6) occasionally, in normal or healthy young individuals .

In long continued pulmonary hypertension, failure of the right ventricle may ultimately supervene, and result in progressive diminution in intensity of the previously accentuated second sound

Accentuation of *both components* of the second sound may be due to (1) extrinsic causes such as a thin chest wall, (2) retraction of lung tissue normally covering the heart, or (3) the coexistence of systemic and pulmonary hypertension

A study of the *comparative intensity* of the second sound in the aortic and pulmonary areas is of some value in diagnosis and follow-up In high blood pressure and in normal elderly subjects, the second sound is louder in the aortic area than in the pulmonary, a contrary finding, in such cases, is almost diagnostic of failure of the left ventricle With recovery from the failure, the normal relationship of the two sounds is again restored

Occasionally, as in children, the aortic component may be better heard in the pulmonary area and the pulmonary one in the aortic area, resulting in errors of diagnosis It is therefore more logical to determine which of the two valve closure sounds is accentuated, rather than evaluate the comparative intensity of the second sound in the aortic and pulmonary areas This is usually done on the basis of the following features (a) the aortic component usually occurs earlier than the pulmonary one, (b) palpability of the second sound in the pulmonary or aortic area may afford a clue to which of the two components is accentuated, (c) the presence of an ejection click in one or other area may afford an important clue, (d) accentuation of a component is usually more obvious in the corresponding auscultatory area (although exceptions to this rule do occur from time to time), (e) on the basis of associated clinical and instrumental findings

**DECREASED INTENSITY** This may either affect one of its two components or both

Attenuation of the *aortic component* may be due to (1) aortic stenosis, with markedly thickened, calcified or immobile valve cusps in which case the second sound may even be absent, (2) aortic regurgitation, (3) systemic hypotension from any cause, (4) left ventricular failure

Attenuation of the *pulmonary component* may be due to infundibular or valvular pulmonary stenosis, which is usually congenital

Attenuation of *both components* may be due to extrinsic causes such as thick chest wall or emphysematous lung

**ABSENT OR MASKED SECOND SOUND** The second sound may not be heard, in one or more areas, in case of (1) aortic regurgitation when masked by the early diastolic murmur, (2) premature beats or atrial fibrillation, where some of the beats may not be strong enough to open the aortic or pulmonary valves, and (3) severe aortic or pulmonary stenosis, where closure of the valve is seriously interfered with

**WIDE SPLITTING ( $A_2$  before  $P_2$ )** Wide or pathological splitting of the second sound (Fig 9 18) depends on an abnormally wide separation of the aortic and pulmonary components of the sound. Although usually due to a lag or delay in the appearance of the pulmonary component, a wide splitting may be due at times to a premature or early appearance of the aortic component. In either case, splitting of the second sound is wide enough to be obvious during expiration

Wide splitting of the second sound may be due to **A Right ventricular abnormality** (1) Complete right bundle branch block. There is a delay in the pulmonary valve closure in such cases because due to delayed electrical activation of the right ventricle the whole right ventricular systole is late. In case of incomplete right bundle branch block or congestive heart failure, this widening of the "split" during inspiration is frequently absent. Splitting is much less obvious, absent or paradoxical in left bundle branch block, because of (a) delay of the aortic component, which normally precedes the pulmonary one, thus tending to narrow or "reverse" the split and (b) a preponderance of elderly subjects, with less likelihood of exhibiting splitting. (2) Atrial septal defect, with left to right shunt at atrial level. The pulmonary valve closure is delayed in such cases because of (a) increased stroke volume of the right ventricle resulting in a prolongation of right ventricular ejection or emptying time, (b) infundibular hypertrophy, and (c) associated incomplete right bundle branch block. The second sound in atrial septal defect is characteristically *wide and fixed*, there being little or no widening of the split during inspiration, because of the right ventricle being already fully loaded or overfilled during expiration by the left to right shunt. (3) Anomalous pulmonary venous return. The masked stroke volume of the right ventricle

due to left to right shunt, as in the case of atrial septal defect, leads to delayed closure of the pulmonary component (4) Pulmonary stenosis with intact ventricular septum due to prolonged right ventricular ejection In valvular or infundibular stenosis the pulmonary component (when audible) tends to be delayed, resulting in a wide splitting of the second sound, the time interval between the two components being proportional to the degree or severity of the stenosis (pressure gradient across the valve) Unfortunately, the value of this sign is often lost clinically because of (a) the masking of the aortic component by the loud murmur and (b) the softness or absence of the pulmonary component In valvular pulmonary stenosis, the degree of widening of the split and duration of the loud ejection murmur are proportional to the degree of stenosis In mild to moderate stenosis, the murmur being short, the wide splitting of the second sound is clearly audible and may simulate that of atrial septal defect In case of severe stenosis, the murmur may be long enough to envelop and drown the earlier aortic component, the soft and delayed pulmonary component alone being heard after a prolonged murmur In such cases, the split, even though unduly wide (as much as 0.16 second in some cases) may be missed completely Graphic registration of the sounds is valuable not only for revealing the wide splitting but also for assessing the degree of severity of the stenosis In Fallot's tetralogy the pulmonary component becomes inaudible Hence audibility of this component favours the diagnosis of pulmonary stenosis with an intact ventricular septum (5) Artificial supraventricular stenosis following banding of pulmonary artery for VSD with large left to right shunt due to prolonged RV systole (6) Pulmonary artery branch stenosis (7) RV failure or disease (7) Pulmonary embolism (sometimes) (8) Left ventricular pacing or ectopic due to delay in onset of electrical activation of RV

**B Left ventricular abnormality** (1) Ventricular septal defect (with L to R shunt) With small defect and low pulmonary vascular resistance the split is often wide and widens further on inspiration The reasons are (a) early aortic valve closure ( $A_2$ ) because of short isometric time of diastolic overload of LV and diminished resistance to LV outflow, (b) delayed pulmonary valve closure ( $P_2$ ) from systolic overloading of RV and delayed onset of activation of RV (2) Mitral regurgitation Here  $A_2$  is early because of diminished resistance to LV outflow and short ejection time (This may disappear when LV is badly affected)

**FIXED SPLITTING** Failure of the two components of the second sound to separate during inspiration is termed fixed splitting It may be due to (1) Atrial septal defect because the RV is already fully loaded or overfilled during inspiration by the left to right shunt Narrowing or disappearance of this sign in such a case suggests reversal of the shunt with pulmonary hypertension and inoperability (2) Anomalous pulmonary venous drainage with intact atrial septum because of delayed closure of  $P_2$  as in atrial septal defect (3)

Right ventricular disease since the R V is not able to increase the stroke volume during inspiration (4) Ventricular septal defect if significantly large, the discrepancy in pressure between the two ventricles is not large so that inspiration affects both ventricles equally, and also in such patients the pulmonary vascular resistance is sufficiently high to greatly reduce the left to right shunt so that volume capacity and duration of systole of both ventricles is then similar and  $A_2$  and  $P_2$  are fixed

*Split second sound at apex* Normally, the inspiratory splitting of the second sound may be heard (besides the pulmonary area) at times over the aortic or left parasternal area but never over the mitral area or apex Splitting of the second sound over the mitral area is due to (1) Pulmonary hypertension The pulmonary component from a forcible valve closure in such cases may be loud enough to be heard at the apex, a most useful sign (2) When the apex beat is formed by a dilated right ventricle instead of the normal left ventricle

*Mistaken diagnosis of abnormal splitting of second sound* may be due to (1) Hurried breathing in children allowing less variation in R V stroke volume (2) Ventricular ectopic beats if frequent (3) Right bundle branch block especially with varying stroke volume as in atrial fibrillation causing further increase in wide splitting on inspiration (4) Late systolic click preceding  $A_2$  or opening snap following  $A_2$  may be mistaken for fixed splitting of second sound

**PARADOXICAL SPLITTING (REVERSED SPLITTING)** This phenomenon occurs when pulmonary valve closure ( $P_2$ ) precedes aortic valve closure ( $A_2$ ), a reversal of the normal state of affairs Thus there is audible splitting of the second sound on expiration During inspiration  $P_2$  moves in the usual way but towards  $A_2$  instead of away from it resulting in a single sound (Fig 9 18) This is the reverse of normal, hence the term reversed or paradoxical splitting

Reversed splitting may be due to **A Electrical LV delay** (1) Complete left bundle branch block is the common cause Aortic valve closure is said to be delayed in such cases not because of a delayed onset of left ventricular systole but because of a slower spread of conduction The splitting becomes more evident during expiration, when right ventricular systole is shortest, resulting in a premature appearance of the pulmonary component (2) Wolf-Parkinson-White (WPW) syndrome Here early activation of R V may result in only slight precedence of  $P_2$  on expiration but normal splitting ( $P_2$  lost) on inspiration (3) Right ventricular ectopic beat or pacing

**B Mechanical delay** (1) Aortic stenosis Because of prolongation of left ventricular systole or emptying time, in severe cases of obstruction to L V outflow at valvular or subvalvular level, the aortic component is delayed and follows the pulmonary During inspiration, the pulmonary component is delayed and the "split" becomes narrower instead of wider This sign is not

very helpful in practice, because of masking of the earlier pulmonary component in many such cases by a loud systolic murmur, resulting in a "pure" or single second sound. In milder cases of aortic stenosis, splitting of the second sound may be "normal" instead of "paradoxical" provided the aortic component is audible (2) Aorto-pulmonary shunt e.g. patent ductus arteriosus. Large left to right shunt leads to prolongation of left ventricular systole from overloading (3) Myocardial disease, ischaemia or infarction (4) Systolic hypertension. Here the split is physiological on inspiration but reversed on expiration (5) Severe aortic regurgitation with an increased stroke volume

**SINGLE SECOND SOUND** The second heart sound will appear single if the two components are separated by interval of less than 0.03 seconds. This is not uncommon in normal individuals over the age of 50 years. Pathological causes of a single sound are—(1) *Changes in  $P_2$*  ( $P_2$  not detected) (a) Diminution in intensity of  $P_2$  as in Tetralogy of Fallot, tricuspid atresia, pulmonary atresia, pulmonary stenosis, transposition of great vessels (b) Distant heart sounds because of hyperinflation of lungs (c)  $P_2$  synchronous with  $A_2$ , Large VSD or single ventricle, some cases of aortic stenosis (d) Masked by systolic murmur as in aortic stenosis (2) *Changes in  $A_2$*  ( $A_2$  not detectable) (a) Diminution in intensity. Calcific aortic stenosis (b) Synchronous with  $P_2$ , VSD with Eisenmenger syndrome, or single ventricle (c) Masked by loud ejection systolic murmur as in pulmonary stenosis or loud pansystolic murmur as in mitral regurgitation or VSD (at apex but not at base) (c) Presence of only one semilunar valve as in truncus arteriosus

#### Abnormal and extra sounds (Fig 9.19) ✓

**Third heart sound ( $S_3$ )** The third heart sound is commonly audible in normal children and adults up to the age of 40 years. After this age it is rare to hear the third sound probably because of increased viscosity of ventricular wall preventing very rapid filling. The presence of a third sound gives rise to triple rhythm and it is advisable not to use this term to include splitting of heart sounds, clicks or extracardiac sounds. Triple rhythm may be physiological or may be a sign of serious heart disease. Its significance can be determined by consideration of associated symptoms and signs (by the company it keeps)

**ABNORMAL OR PATHOLOGICAL THIRD HEART SOUND (ventricular gallop)** The pathological third sound can be differentiated from the normal or physiological third sound by (a) left or right ventricular enlargement, heart disease, or cardiac failure, (b) the sound being louder in intensity than the normal third sound, (c) age over 40 years, (d) presence of palpable diastolic thrust, (d) maximum intensity over the cardiac apex in the case of left ventricular, and near the lower end of sternum in case of right ventricular gallop, (e) diminution in intensity by sitting or standing and accentuation by brief exercise

*Mechanism* The *classical* view that sudden stretching of the ventricular wall through overdistension (either through excessive volume of blood or a diseased myocardium with reduced compliance), results in an audible sound because of vibration of the muscle wall (not unlike the sound produced by "wind when filling an empty sail"), is being abandoned of late in favour of a *valvular* hypothesis. Vigorous elongation of the left ventricle (during the phase of rapid ventricular filling) leads to ascent of the mitral annulus fibrosus, with sudden tautening or "tensing" of the mitral leaflets and chordae tendinae resulting in an audible third sound. Accentuation of the latter occurs, when ventricular filling is prolonged beyond the third sound, because of overfilled left atrium and increased atrioventricular pressure gradient. According to this theory, prolonged ventricular filling (through a *relatively* stenotic orifice) from an overfilled left atrium, leads to prolongation and accentuation of the third sound (or a ventricular gallop sound). Rest, prolonged standing and other manoeuvres capable of altering the venous return to the heart, by altering the state of ventricular distension, bring about alterations in the incidence, intensity and timing of ventricular gallops. The greater the distension of the ventricle, the more the tensing of the cusps and tendinae and greater the intensity of the pathological third sound. Of late, the *valvular* theory of ventricular gallop has more or less completely superseded the old *muscular* theory of mechanism of diastolic gallop.

An abnormal third sound may be due to (1) Ventricular failure causing raised atrial pressure — (a) *A left sided third sound* has its greatest value as a sign of abnormal L V function with L V failure, myocardial ischaemia or infarction, hypertension or cardiomyopathy. It is common with severe mitral incompetence. (b) *A right sided third sound* is common with massive pulmonary embolism and certain forms of cardiomyopathy. (2) Constrictive pericarditis (pericardial knock sound). (3) Mitral regurgitation and rarely tricuspid regurgitation. When judging the relative preponderance of mitral regurgitation or stenosis, a left sided third sound excludes dominant stenosis.

**Fourth heart sound (Atrial sound)** In normal subjects this sound is seldom if ever audible though it can be recorded in a sound tracing. Audible atrial sound (as in complete heart block) gives rise to triple rhythm. When  $S_3$  and  $S_4$  co-exist, they produce a quadruple rhythm.

**PATHOLOGICAL FOURTH HEART SOUND** An audible fourth sound is a useful sign of ventricular abnormality. Its site of origin whether the left or right atrium can be identified by other physical signs.

*Mechanism* With the aid of atrial electrocardiogram, the atrial gallop is shown to be due to certain late vibrations which occur during the earliest part of ventricular systole. The response of this sound to respiration, postural changes, Valsalva manoeuvre, carotid sinus stimulation and alterations of blood pressure proves it to be a ventricular filling sound or a ventricular filling

event, incidental to atrial contraction and caused by overloading of the ventricle during systole

**Causes** A *left atrial* (left sided)  $S_4$  is usually best heard at the apex, with the patient turned to the left and is louder during inspiration. It occurs in (a) disorders characterised by decreased compliance of the left ventricle such as systemic hypertension, aortic stenosis or regurgitation and cardiomyopathy (b) Acute myocardial infarction (c) Angina pectoris. A fourth sound may appear transiently during an attack of angina pectoris and is a useful sign of ischaemic heart disease particularly when the electrocardiogram is normal (d) Prolonged atrio-ventricular conduction (e) Hyperkinetic circulatory states such as anaemia or hyperthyroidism (f) Acute mitral regurgitation from ruptured chordae tendinae. A *right atrial* (right sided)  $S_4$  is best heard along the left lower sternal border and tends to be louder during inspiration. It results from either decreased compliance of the right ventricle or from increased resistance to right ventricular filling. It may occur with (a) Pulmonary hypertension (b) Pulmonary stenosis (c) Cardiomyopathy (d) After massive pulmonary embolism

**GALLOP RHYTHM** (Fig 9 19) Certain varieties of "triple rhythm" have been labelled "gallop rhythms". The presence of an accentuated or pathological third heart sound, an atrial sound and a fusion of the two, during ventricular diastole, have been arbitrarily labelled as (1) *ventricular* (or protodiastolic), (2) *atrial* (or presystolic) and (3) *summation gallop* sounds respectively. The combination of the two normal heart sounds with a gallop sound when associated with tachycardia, being suggestive of the "gallop" or "canter" of a horse, is referred to as gallop rhythm. Because of its frequent association with myocardial failure this sound is often of serious prognostic significance and the louder the added sound the more serious the outlook.

**SUMMATION GALLOP AND QUADRUPLE RHYTHM** In states of tachycardia or less often with prolonged P-R interval (A-V conduction time) there is superimposition of atrial and ventricular added sounds giving rise to a 'summation gallop'. It is easily detected because of loudness of the sound and is probably the variety of pathological triple rhythm most frequently recognised. It may be possible to demonstrate the fusion or summation of two added sounds (quadruple rhythm) by slowing the heart with carotid sinus stimulation.

**Pericardial knock** An extra sound in early diastole and due to rapid ventricular filling is frequently heard in constrictive pericarditis. It results from two factors namely combination of rapid flow from raised venous pressure and the abrupt limitation of ventricular filling produced by the fibrous or calcified pericardium. At times the sound is loud and high pitched and may resemble an opening snap. As compared to other extra sounds it occurs a little earlier after the second sound.

**Ventricular knock.** A loud sound may be audible in cases of severe aortic regurgitation or mitral regurgitation. It occurs at the same time as the ven-

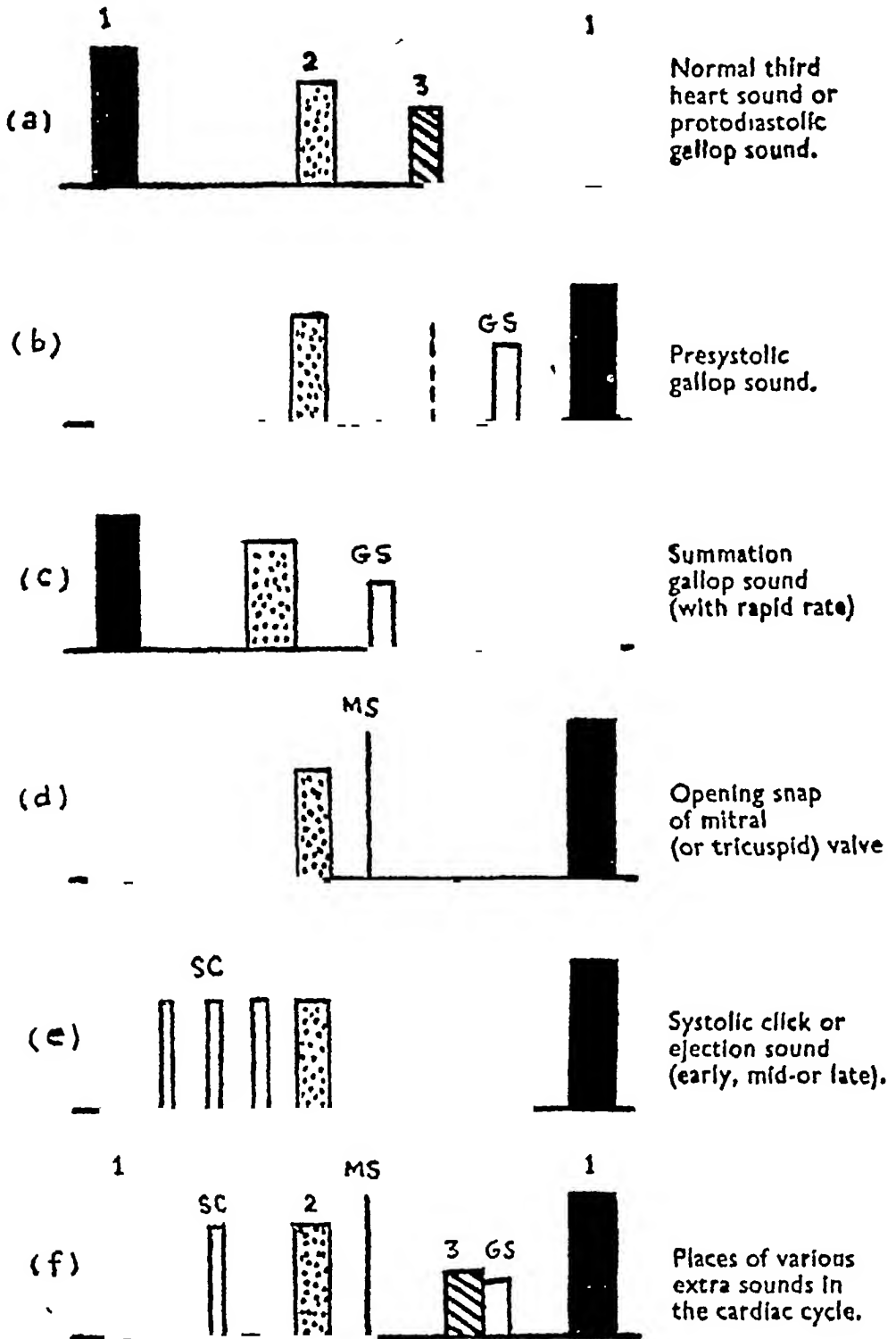


Fig 9 19. Triple heart rhythm Varieties of abnormal and extra heart sounds in the cardiac cycle



tricular gallop and is probably produced when the diastolic movement of the heart during rapid filling causes the heart to strike the chest wall

Snap sounds (Early diastolic valve opening sounds)

A Opening snap due to A-V valve stenosis with mobile valve

1 OPENING SNAP OF THE MITRAL VALVE (Mitral snap) Midway between the second and third heart sounds (about 0.09 to 0.13 second after the onset of the aortic or early component of the second sound) in over 90 per cent of cases of "pure" or "dominant" mitral stenosis secondary to acquired rheumatic valvular heart disease, is usually heard a characteristic high-pitched, loud, snapping or clicking, sharp sound

The *mechanism* of production of the opening snap is similar to that of the accentuated first heart sound of mitral stenosis, there being a close correlation between these two sounds. The opening snap (O S) is due to a sudden or sharp "tensing" or "buckling" of the cusps of the mitral valve as it tries to open during early diastole (at the end of the isometric relaxation phase), when the left atrial pressure exceeds that of the left ventricle. The sound is caused by a sudden cessation of the opening movement of the stenotic, but somewhat flexible and fibrosed, mitral valve. Vibration of the large "anterior" leaflet is mainly responsible for the snap sound, other factors contributing to its intensity being (a) moderate calcification and fibrosis of the mitral valve. Severe calcification, however, abolishes the sound, because of lack of pliability and mobility of the valve. (b) left atrial pressure. The opening snap is of great diagnostic value in mitral stenosis, especially when the characteristic mid-diastolic and presystolic murmurs happen to be absent, or if mitral flow is small due to pulmonary hypertension, right heart failure or tricuspid regurgitation.

The opening snap may be *absent*, in occasional cases of mitral stenosis, when (1) the duration of preceding diastole, which if long, allows a high L.A. pressure to fall and the O S may be absent possibly because of the slowing of the rate of movement of the valve displacement into the L.V. owing to ventricular distensibility or aortic regurgitant stream. (2) The stenotic process is very early or mild, (3) the valve is markedly calcified or fibrosed, with lack of mobility or pliability of the cusps. (4) mitral regurgitation of moderate degree is associated with the stenosis. In mitral valve disease with dominant or pure mitral regurgitation, the opening snap is usually absent, because of (a) deformity and rigidity of the mitral leaflets, and (b) lack of a significant pressure-gradient (between the left atrium and ventricle) at the end of diastole. Even with "mild", superadded regurgitation, the opening snap may be prevented in otherwise typical cases of mitral stenosis, because of lack of movement of the anterior leaflet of the valve, secondary to impingement on it of the regurgitant stream of blood. Occasionally, in cases of mitral regurgitation (even when severe), the opening snap may persist when a mobile and pliable anterior

valve cusp is associated with a severely affected and rigid posterior one (5) A high pulmonary vascular resistance, this may prevent the occurrence of a "snap" sound in mitral stenosis, for some obscure reason, (6) fusion or matting of the chordae tendinae and papillary muscles of the left ventricle In VSD with PDA and left to right shunt a mitral snap may be recorded but is seldom obvious

*Characteristics* of an opening snap are (1) The sharp and snappy, clicking character of the sound, similar to the "clicks" heard over the telephone, (2) the site of maximum intensity of the sound, which is along the left sternal border at the level of the fourth interspace, a site midway between that of a split second sound (pulmonary area) and of a third heart sound (apical area), (3) transmission over a wide area, particularly over the basal areas and supra-sternal notch (because of transmission and registrability of the sound within the left atrial cavity), (4) accentuation by exercise, (5) heard best with the diaphragm of the stethoscope, (6) shows a wider separation than usual from the second sound, in the standing position, (7) deep inspiration fails to alter its position in the cardiac cycle (unlike a split second sound), (8) persistence, despite the presence of atrial fibrillation, (9) persistence, as a rule, after mitral valvotomy, in both calcified and uncalcified types of stenosis (because of subvalvular fusion), and (10) its association with an accentuated first sound

The "2-O S interval" (interval between the onset of the second heart sound and the opening snap) is a good guide to the "severity" of mitral stenosis, although dependant (to some extent) on other factors also, such as left atrial pressure, systemic arterial blood pressure and length of the preceding cardiac cycle With "slight" stenosis and low atrial pressure, the 2-O S interval is usually 0.10 to 0.14 second, with "tight" stenosis alone about 0.04 to 0.05 second, and with tight stenosis and high left atrial pressure the interval is below 0.04 second Besides mitral stenosis, which shortens the 2-O S interval (the more severe the stenosis, the shorter the 2-O S), a high left atrial pressure (as in atrial myxoma) or a short preceding cardiac cycle (as in tachycardia, atrial fibrillation or after exercise) may also shorten the 2-O S interval

When the opening snap is present in case of mitral stenosis with regurgitation, the 2-O S interval is usually short, because the regurgitant stream (by raising the left atrial pressure and volume at the end of ventricular systole) results in an earlier opening of the mitral valve Factors conducive to long 2-O S intervals in mitral stenosis are bradycardia and systemic hypertension, where delayed opening of the mitral valve is due to an unduly high left ventricular pressure (during the interval between the closing and opening of the mitral valve)

The opening snap must be clearly distinguished from a split second sound and ventricular gallop or third heart sound, in cases of mitral stenosis, because of the different clinical significance of the signs The interval between the

two components of a *split second* sound, besides being much shorter than the 2-O S interval, tends to increase (unlike the O S, which remains unaltered) on inspiration, but is unaffected by standing (a manoeuvre that increases the 2-O S interval). The *third* or *gallop* sound differs from an "opening snap" in being low-pitched, better heard with the bell-stethoscope and arising much later in diastole (0.12 to 0.24 second after the second sound). Whilst an opening snap suggests preponderant stenosis and is against free mitral regurgitation, a gallop sound suggests dominant regurgitation and is most unusual in stenosis (being dependant on rapid ventricular filling, a condition not possible in tight stenosis of the mitral valve).

*Other causes of opening snap* Besides acquired rheumatic heart disease with dominant mitral stenosis, other conditions, likely to give rise to this sound from time to time, are (1) Congenital mitral stenosis, a very rare malformation of the heart which may be associated with a snap sound. (2) Mitral regurgitation, when associated with a rigid posterior but normal anterior valve leaflet. (3) Primary subendocardial fibroelastosis, when associated with stiffness and fibrosis of the mitral valve. (4) Left atrial myxoma (in 10 per cent of cases only) because of the markedly raised left atrial pressure. The snap is usually soft and late and the intensity of the snap varies greatly with the position of the patient. (5) Excessive blood-flow through the mitral orifice, as in case of large ventricular septal defects or patent ductus arteriosus. (6) Gargoylism, with involvement of the heart as a rare complication.

**OPENING SNAP OF THE TRICUSPID VALVE (TRICUSPID SNAP)** An opening snap of the tricuspid valve is much less common (although similar in character) than the mitral snap. It is seldom heard because of the almost invariable presence of a mitral stenosis and a mitral snap. It is usually best heard at the lower end of the sternum or along its right border, and is frequently associated with a "mitral snap". Since the latter is usually well heard all over the precordium and is far more common, the diagnosis of a tricuspid snap is frequently difficult or unjustified. It is due usually to (a) tricuspid stenosis (frequently in association with mitral stenosis and a mitral snap) (b) atrial septal defect with left to right shunt, or (c) chronic constrictive pericarditis.

#### B A-V abnormality without stenosis.

In Ebstein's anomaly the large anterior cusp often gives rise to a loud diastolic opening sound which is closely associated with a loud diastolic tricuspid closure sound. Because of several sounds and murmurs, auscultation is not easy but the combination of wide splitting of the second sound and a clicky diastolic sound louder during inspiration in a patient who has central cyanosis is virtually diagnostic of Ebstein's anomaly.

#### Systolic ejection sounds or clicks.

**EARLY SYSTOLIC EJECTION SOUNDS** (sometimes termed the opening snap of the semilunar valve) These are high-pitched, click-like sounds synchronous

with ventricular systole and in most cases seem to arise from the movements of the valve cusps. However, a click may also be heard with dilatation of the ascending aorta or main pulmonary artery and a normal valve. The added sound is high-pitched, arises 0.08-0.10 seconds after the onset of the first sound and may be aortic or pulmonary in origin. It is often present in elderly patients and is of no significance. A click is of value in determining whether the obstruction to ventricular outflow is at valvar level, though it may be absent due to rigidity of the cusps.

*Mechanism* The onset of ejection into the aorta and pulmonary artery is associated with setting up of vibrations which become accentuated to form ejection sounds. With simultaneous study of cine-aortograms, sounds and pressure pulses, it has been shown that the upward movement of fused valves e.g. in aortic stenosis halting of the upward movement coincides with the ejection sound. The same mechanism causes the ejection sound in pulmonary stenosis. In case of dilatation of aorta and pulmonary artery the ejection sound appears to have the same relation to the termination of the opening movement of the valve.

**AORTIC EJECTION SOUND** It is heard in both aortic and pulmonary areas and transmitted to the apex, and does not vary with respiration. It is heard in (1) Aortic valve disease (a) Aortic stenosis of valvar type with mobile valve. It does not occur in infundibular stenosis or obstructive cardiomyopathy. (b) Bicuspid aortic valve. (c) Aortic regurgitation. In minimal aortic leak the presence of ejection sound necessitates careful auscultation for a diastolic murmur. (2) Dilatation of ascending aorta from hypertension, aortic aneurysm, dissection, coarctation, atheroma, syphilitic aortitis, severe tetralogy of Fallot or pulmonary atresia.

**PULMONARY EJECTION SOUND** It is best heard in second left space, not transmitted to the apex, and occurs earlier than the aortic ejection sound. The causes are (1) Pulmonary valve disease. In pulmonary stenosis the ejection click varies with respiration frequently disappearing on inspiration. An absent ejection sound in pulmonary stenosis suggests an infundibular stenosis or that the cusps are unduly thick with possibility of difficulty during surgery. (2) Pulmonary artery dilatation (a) Pulmonary hypertension. Here there is little respiratory variation. (b) Idiopathic dilatation of pulmonary artery.

**MID OR LATE SYSTOLIC (POST-EJECTION) CLICKS** These are best heard at the apex or in the midprecordium and may show respiratory or positional variation in intensity or even in timing from beat to beat. They are usually single but at times two or more sounds may be heard. Causes (a) These sounds are in majority of cases benign and of no significance and occur after the onset of ejection. (b) Systolic clicks associated with mid- or late systolic murmurs are indicative of non-rheumatic mitral regurgitation, and have been shown to be due to unusual ballooning of the mitral cusps into the left atrium.

*Effect of Valsalva and other manoeuvres on heart sounds and clicks* (a) With *Valsalva manoeuvre* the systolic click in the systolic click late-systolic murmur syndrome moves towards the first sound. The two components of the second heart sound become fused except in ASD and RBBB. Diastolic gallop sounds fade or disappear. (b) *Isonitric hand grip* may bring out or accentuate gallop sounds in ischaemic heart disease or congestive cardiomyopathy. (c) *Carotid sinus massage*. With a rapid heart rate a gallop sound occurring in mid-systole (summation gallop) may be difficult to identify as fourth or third heart sound. Pressure on carotid sinus by slowing the pulse and producing quadruple rhythm can enable one to identify the closer proximity of the sound to the first or second sound.

**Extra cardiac systolic sounds** There are several varieties of extra sounds or clicks of extracardiac origin which may be audible over the heart during mid- or late-systole. Usually ascribed to the movement of the heart in relation to other mediastinal structures, they may be due to (1) Left-sided pneumothorax. This may cause a clicking sound, at times loud enough to be heard at a distance and affected considerably by respiration and change of posture. The systolic click which is usually evanescent, is particularly common in the later stages of a resolving left pneumothorax. (2) Pleuroperecardial adhesions. (3) Spontaneous mediastinal emphysema (with peculiar "crunching sounds" synchronous with the heart). (4) Emphysematous bleb or bulla, close to the heart. (5) Funnel-chest, where a substernal or xiphisternal crunch (a superficial crunching sound, probably arising in the joint of the seventh costal cartilage with the sternum) is sometimes audible. Extra systolic sounds are entirely harmless, occur much later in systole than ejection sounds and are always extracardiac in origin. (6) Sounds produced by cardiac pacemakers. The sound heard during presystole results from contraction of skeletal muscle and is not of cardiac origin. (7) Artificial valve sounds. These vary with the type of artificial or prosthetic valve implanted and may be opening or closing sounds.

**Extraneous adventitious sounds.** During auscultation certain extraneous or adventitious sounds may appear and interfere with a proper hearing or interpretation of heart sounds and murmurs. The most important of these are (1) extraneous noises or sounds from outside. (2) pectoral muscle sounds usually rumbling or roaring, dull sounds but, occasionally soft and rale-like, (3) hairy crepitations (due to friction of the chestpiece with the hair on the chest-wall and easily removable by moistening or shaving of the hair).

**Vascular sounds** *Heart sounds* One or both of the heart sounds may be audible over the carotid and subclavian arteries in normal subjects, especially on the right side.

*Arterial sounds* A systolic sound may occasionally be heard over the femoral artery, even without compression of the artery. A marked accentuation of the systolic sound over the femoral or brachial artery, resulting in the

so-called "pistol shot sound", is common in aortic regurgitation and in any condition, in which the pulse pressure is high and the diastolic pressure low. The pistol sound is probably due to vibrations of high frequency, caused by a sudden elevation of pressure within the vessel. Occasionally, a double sound (Traube's double tone) is heard in aortic incompetence, instead of the single pistol shot.

## CARDIOVASCULAR MURMURS

**Definition** A cardiovascular murmur may be defined as a prolonged series of auditory sounds or vibrations of varying loudness (intensity), frequency (pitch), character or quality, duration and configuration and caused by the vibrations of the valves or walls of the heart or great vessels.

Although a murmur seldom sounds like a "murmur" in the literal sense of the word, being more often blowing or rumbling in character, the word murmur has enjoyed universal acceptance for so long that it is difficult to displace it.

Murmurs, also called "bruits", have been greatly overrated in the past, and later grossly underrated, as evidence of heart disease. In conjunction with other signs and a proper history, murmurs may offer information of great diagnostic or prognostic value.

**Noteworthy features** Certain features of general interest about murmurs are worth noting. (1) The presence of a murmur does not necessarily imply a diseased heart, for instance, an insignificant or innocent murmur is usually associated with a normal heart. (2) Serious heart disease (e.g. myocardial infarction, angina pectoris or congenital heart disease) may exist in the absence of all murmurs. (3) The intensity of a murmur is usually no indication of the degree of the causative lesion. As a matter of fact, in mitral stenosis, aortic stenosis or ventricular septal defect, progression of the disease or advent of heart failure may cause a murmur to become fainter or even disappear. (4) The length or duration of a murmur is often of greater significance in diagnosis and prognosis (as in the case of mitral valve disease) than its intensity or loudness. (5) A murmur heard best over the heart need not necessarily be of cardiac origin, it may be extracardiac in origin. (6) A very loud murmur or "cooing" murmur is usually indicative of organic heart disease, a musical or rasping murmur may be due to eventration of a valve cusp or torn chorda tendinae. (7) Whilst a diastolic murmur suggests organic heart disease, a systolic murmur may be insignificant or innocent. (8) Whilst an innocent murmur is usually profoundly altered by respiration or change of posture, a significant murmur is only slightly affected or not at all. (9) A so-called stenotic or regurgitant murmur need not be valvular in origin, it may be due to a relative or functional stenosis or regurgitation, which may be more important or serious than the valvular defect. (10) Whilst post-natal or acquired murmurs tend to originate in the valves of the left side of the heart (mitral and aortic valves), congenital murmurs usually arise in the pulmonary valve in the right side of the heart.

**Mechanism of production** Three main theories, viz. the turbulence theory, the cavitation theory and the vortex-shedding theory, have been put forward in attempts to explain the physio-pathological mechanism of murmur production.

(1) *Turbulence theory* According to the classical and once widely accepted theory of "turbulence", the conversion of the normal "laminar" flow of blood into a "turbulent" one

results in a murmurish sound or murmur. Turbulence of a "random flow of fluid particles, in terms of direction and velocity", was said to occur, when  $R$  or the Reynold number (i.e. diameter of channel  $\times$  stream velocity  $\div$  viscosity) exceeded the critical level of 2000. Being applicable to both obstructive (stenotic) and non obstructive conditions (such as anaemias and thyrotoxicosis), this theory of murmur production held sway for many years. In the light, however, of the following facts, the turbulence theory appears no longer tenable: (a) Aerodynamic research (as in case of jet engines) has proved the importance of high velocities of flow (approximately the speed of sound in any given medium) in the generation of sound phenomena. Such high speeds of flow can never be approximated or attained by the slow moving blood particles of the circulatory system. (b) The Reynold number of pulsating blood within the major arterial trunks is in the region of 1000 (or well below the critical level of 2000). If "turbulence" was possible at such low levels (say 1000), then normal individuals would all be subjects of long or pansystolic murmurs over their arterial trunks. (c) If "turbulence" was truly concerned in the production of murmurs, then all murmurs would have to be of the same pitch. The wide differences in pitch displayed in practice by various murmurs is against such a theory.

(2) The *cavitation theory* ascribes the production of murmurs to the phenomenon of "cavitation" or rapid formation and breakdown of "bubbles" of blood (associated with loud crackling noises) during their flow downstream. This is said to occur when too rapid a flow of blood (through an orifice) results in a local lowering of pressure (beyond the orifice) to a level below the vapour pressure of blood. Cavitation has been experimentally induced and studied in various models, employing both sustained and pulsatile flows of blood. As an explanation of human murmurs, this theory is unacceptable for two reasons: (a) "crackling" noises, produced during experimentally induced cavitation, are much louder and easily audible without a stethoscope, (b) pressures of about 350 mm Hg or much higher than can be attained in human beings, are required for inducing "cavitation" in experimental models.

(3) The *vortex shedding* theory of aeolian tone formation seems to be the most acceptable explanation for the mechanism of heart murmurs and their variable behaviour under changing haemodynamic conditions. A smooth obstacle or body, such as a cylinder or wire (or valve structure) intercepting the path of any fluid (say blood) leads to the phenomenon, known as "vortex shedding," along its edges. Irrespective of whether vibration of the vessel wall or obstacle (such as a deformed valve cusp) is present or not, pressure oscillations of high magnitude are generated within the fluid medium itself, resulting in an aeolian tone or sound. The frequency and intensity of the tone or sound produced depends on the position of the auscultator in relation to the blood flow, on the velocity of flow (the frequency of tone being proportional to velocity) and on the diameter of the orifice (e.g. stenotic valve). It is possible to explain, in terms of the vortex shedding theory, certain obscure clinical phenomena, such as the presence of high frequency murmurs with slight or minimal stenosis and of low frequency murmurs with moderate or severe stenosis, of high pitched murmurs in regurgitant states, such as aortic insufficiency, the association of a high-pitched systolic murmur at the apex in conjunction with a low pitched murmur over the aortic area in case of uncomplicated aortic stenosis, the occasional persistence of diastolic murmurs in mitral stenosis after radical correction through mitral valvotomy, the high incidence of systolic murmurs in normal individuals after exercise (during fevers or with hyperdynamic circulatory states), and the phenomenon of post stenotic dilatation, distal to obstructive lesions of heart valves. Dilatation of vessels, distal to valve stenoses, are probably due to high magnitude pressure oscillations secondary to the phenomenon of vortex shedding.

Regardless of the exact mechanism of sound formation as related to murmurs, production of murmurs can be attributed to the following factors (Fig 9 20)

(1) Forward flow or rush of blood from a relatively narrow into a wide adjoining part or section of the heart or blood vessel, the flow of blood may be through a narrow, constricted or stenotic valve into a normal-sized heart chamber (stenotic or obstructive murmur) or through a normal valve into a

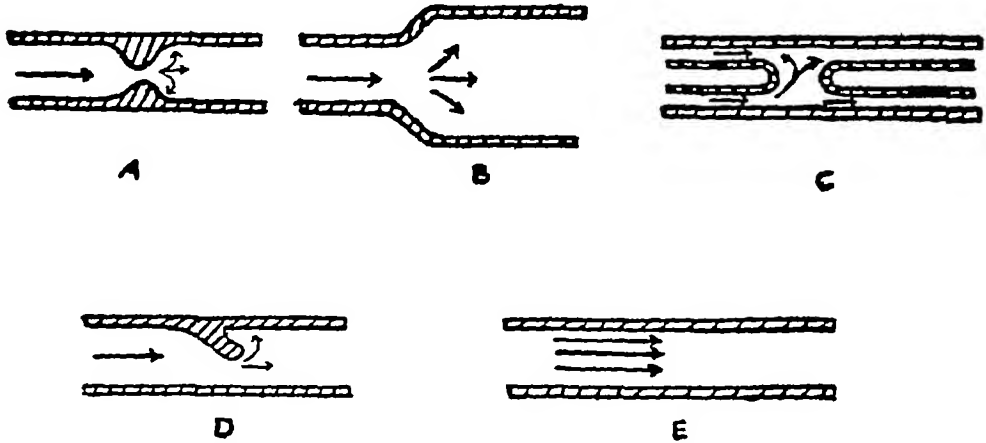


Fig 9.20 Basic mechanism of cardiac murmurs A Forward flow through narrowed valve into normal chamber, B Forward flow through normal valve into dilated chamber or vessel, C Backward flow through regurgitant valve or defect, D Vibration of loose structure or vegetation, E Increased velocity of blood flow (hyperkinetic murmur)

dilated chamber or blood vessel (relative or non-valvular stenosis) The production and intensity of the resultant murmur depend on the degree of difference of calibre of the adjoining parts, the abruptness of the change of calibre, the pressure differential and the rate of flow of blood (2) Backward flow (or jet lesion) of blood through a regurgitant valve (a valve that cannot close and thus allows reflux of blood is said to be regurgitant or insufficient), septal defect or patent ductus (3) Increased velocity or high rate of flow of blood (hyperkinetic murmur) (4) More or less continuous flow through extracardiac and intracardiac shunts and narrowed or altered vessels, and in vascular structures (5) Rarely vibration of a loose structure, such as a torn cusp, chorda tendinae or vegetation in the blood stream (vibrational or string murmur) A combination of these factors may be responsible

The mechanism of production of a murmur may be anomalous and outside the heart and main vessels, as in the case of cardiopulmonary murmurs, or obscure or unexplained in spite of investigations (*agnogenic* murmur)

**Clinical investigation of murmurs** Every murmur, however insignificant, must be subjected to close scrutiny and investigation The following are the most important characteristics or diagnostic features of cardiovascular murmurs, which are routinely investigated

1 **Timing or position in the cardiac cycle** The first and most important characteristic of a murmur is its exact position or place in the cardiac cycle



whether systolic, diastolic or continuous The phase of systole starts with the onset of the first heart sound and ends with the onset of the second Any murmur that occurs during this phase is called *systolic*, when long enough to occupy the whole of systole, it is known as *holosystolic* or *pansystolic* (Fig 9 21) A murmur which starts with the first sound (either accompanying, masking or replacing it) is called *early systolic*, a murmur which starts after the completion of the first sound may be either *mid-* or *late systolic*

From the onset of the second heart sound to the onset of the first is the diastolic phase, it is a longer event than systole and is divided into early, mid- and late diastole A murmur, when occurring early in diastole (just after the second sound), is called *early diastolic*, when occupying the middle of diastole (or midway between the second and first heart sounds) *mid-diastolic*, and when occurring towards the end of diastole or just prior to the first sound is called *late diastolic*, *telediastolic*, *presystolic* or *atriosystolic* (Fig 9 22)

A murmur that continues or extends throughout systole and diastole is known as a *continuous* murmur (Fig 9 23) Systolic and diastolic murmurs when occurring together, give rise to the so-called *seesaw*, *to-and-fro*, bellows, double or combined murmur (Fig 9 24) A systolic murmur that extends through or beyond the second sound into early diastole, or "spills" into diastole, is termed a *systolico-diastolic* murmur

The *timing* of a murmur is usually easy with a regular and slow heart rate, difficult or impossible in a rapidly beating or irregular heart, especially with an inexperienced observer The timing of a murmur depends on a correct identification of the heart sounds by careful auscultation of the heart The less experienced may identify cardiac systole by inspection or palpation of the apex thrust or carotid pulsation, both these events being synchronous with ventricular systole The radial pulse is not suitable for the timing of murmurs because of the time delay or "lag" between cardiac systole and radial pulsation

The identification of the nature and site or mode of production of a murmur frequently depends on the correct timing of the murmur A systolic murmur, which is far more common than diastolic, may be innocent or insignificant A diastolic murmur, on the other hand, is nearly always indicative of valvular or structural heart disease A murmur which masks or replaces a heart sound is of greater significance than one which does not

The identification of a systolic murmur is helped by attention to associated phenomena e.g. early systolic ejection sounds are common with dilatation of aorta or pulmonary artery especially when associated with aortic or pulmonary valve stenosis Midsystolic clicks occurring at the beginning of an apical late systolic murmur usually indicate mitral regurgitation The first sound is soft in mitral regurgitation. Wide splitting of the second sound without respiratory variation suggests that a pulmonary ejection systolic murmur is due to A S D

A single second sound is found in tetralogy of Fallot, tricuspid atresia, transposition of great vessels and truncus arteriosus. A faint delayed closure of pulmonary valve occurs in pulmonary stenosis.

According to its timing or position in the cardiac cycle, a murmur may be described as pansystolic or long systolic, as in the case of mitral or tricuspid regurgitation, late systolic or short systolic, as in the case of an innocent murmur, early diastolic, as in aortic or pulmonary regurgitation, mid-diastolic, as in mitral or tricuspid stenosis, presystolic or late diastolic also as in mitral or tricuspid stenosis, continuous, as in patent ductus or arteriovenous fistula, double murmur, as in the case of the seesaw or to-and-fro murmur of aortic incompetence and stenosis or pulmonary stenosis and incompetence, or as a multiple murmur, when more than two murmurs are combined, as in mitral and aortic valve disease.

**2 Site of maximum intensity.** In the case of any murmur, systolic or diastolic, the exact localization or site of maximum intensity must be determined by careful auscultation, as it may (a) suggest its site or mode of production, as in the case of the mitral stenosis murmur in the apical area, or (b) facilitate its recognition, as in the case of Roger's area at the inner end of the third left interspace.

The localization of a murmur may prove difficult or impossible in case of (a) inconstancy of localization or multiplicity of sites of maximum audibility (e.g. the diastolic murmur of aortic regurgitation, which may be best heard either in the aortic area, at the inner end of the third left interspace or at the apex), and (b) diffuse or widespread murmurs, as in coarctation of the aorta where the murmur is widely heard over the back and front of the chest, and in aortic stenosis, where the murmur is heard all over the chest and up the neck on either side.

**3 Intensity or loudness.** The great majority of murmurs are due to rush of blood, from a narrower into a wider section of the cardiovascular system, causing the formation of "eddies" in the blood stream, which set the walls or valves into vibration, the resultant acoustic phenomenon being called a murmur. The intensity of the murmur will therefore depend on (a) the difference in calibre between the adjoining narrow and wide section of the heart or blood vessel, (b) the abruptness of change of calibre, (c) the pressure differential, (d) the rate of blood flow, and (e) external modifying factors, like the thickness of the intervening chest wall or lung tissue.

Murmurs display wide variations in intensity, from the very faint or hardly audible at one end of the scale to the extremely loud at the other. In a case of aortic stenosis recently observed, the murmur could be heard about four yards away from the patient with the bare ear.

The intensity of a murmur is not always proportional to the degree or importance of the causative lesion or haemo-dynamic disturbance. For instance,

the murmur of a small and insignificant ventricular septal defect or mitral regurgitation may be much louder than that of a large atrial septal defect, where a murmur may even be non-existent. The difference depends on the velocity of the jet (or blood flow through the defect), which is much higher in the former instances and thus productive of a louder murmur. With the progression of a valve lesion the associated murmur may show either an increase or decrease of intensity, whilst the advent of heart failure usually makes the murmur lose its initial loudness. Alterations of intensity are often of importance in the follow-up of cases.

**GRADES OR DEGREES OF INTENSITY** Six different grades of intensity or loudness of systolic murmurs have been described as follows

Grade 1 very faint (or can be heard only with special effort)

Grade 2 faint but can be heard easily

Grade 3 moderately loud

Grade 4 very loud

Grade 5 extremely loud

Grade 6 loudest possible (can be heard with the stethoscope just removed from contact with the chest)

Systolic murmurs of grade 3 or more in intensity are as a rule significant. Systolic thrills are usually associated with grade 4 or louder murmurs. Diastolic murmurs are classified usually into four grades. For the sake of simplicity it may be preferable to classify the murmurs according to intensity into soft, moderately loud and very loud.

**Intensity behaviour of murmurs** A murmur may be of constant intensity throughout its duration, of increasing intensity (*crescendo* type), of decreasing intensity (*decrescendo* or *diminuendo* type) or a combination type (*diminuendo-crescendo* or *crescendo-diminuendo*). A crescendo-decrescendo murmur is sometimes referred to as "diamond-shaped" or as having a "christmas tree" configuration.

**4 Quality or character.** A murmur is seldom "murmurish" in the literal sense of the word. It is more often describable as a blowing, rasping, churning, rumbling, rolling, grating, rough, sawing or musical type of sound. The character or quality of a murmur may be characteristic enough (like the bark of a dog or the "purr" of an engine) to permit an immediate recognition of its true nature.

Of the numerous types of murmurs encountered in practice, the following are some of the most characteristic, being recognizable by their quality or character alone. (a) the rumbling or rolling diastolic murmur of mitral stenosis (aptly compared to the sound of a "wooden cart going over cobblestones"), (b) the continuous, machinery, humming-top or train-in-tunnel murmur (Gibson's murmur) of patent ductus arteriosus, (c) the soft, blowing, "whiff-

like" early diastolic murmur of aortic regurgitation, variously likened to the "sucking of breath", "sound of pouring water" or "the sound of a whispered R", (d) the musical or sea-gull murmur of aortic incompetence, associated either with eversion or retroversion of a valve cusp or a tightly stretched moderator band, (e) the loud, rough, harsh, rasping or grating, high-pitched systolic murmur of aortic stenosis, and (f) the water-wheel murmur of hydro-pneumopericardium, (g) the innocent murmur compared to the noise made by a twang string

As a rule, *regurgitant murmurs*, caused by "leaking back" of blood, are of *blowing* or "bellows" type (*bruit de soufflet*), whilst obstructive or *stenotic murmurs* are *harsh, rasping* or *churning* in character, probably because of increased resistance to the flow of blood

Sudden changes in character or intensity may be displayed, from day to day or temporarily, by certain murmurs, especially in cases of bacterial endocarditis, and ruptured cusp or chorda tendinae. The murmurs are referred to as *metamorphosing* (or changing) murmurs

**5 Extent or area of distribution** A murmur may be either localized or confined to a small area as in mitral stenosis, diffuse as in mitral incompetence, or widely diffuse as in aortic stenosis and coarctation of the aorta. The extent or "auditory field" of a murmur may be wide enough to involve even the extremities as in aortic stenosis, the murmur being frequently audible over the olecranon processes

When a murmur is loudly heard over two (or more) valve areas, it is of importance to decide (1) whether the same murmur is audible over both the valve areas, or (2) whether there are two distinct murmurs present. The question is usually settled by tracing the murmur inch by inch, from one valve area to the other, noting the intensity and character of the murmur during the process. In case of gradual increase or decrease in intensity from one area to the other (intensity pattern of "triangular type") and with no change in quality or character, the same murmur is probably heard at both the areas. On the other hand, if the intensity, when traced from one area to the other, shows a decrease to a midway point followed by an increase (intensity pattern of "hour-glass type"), and if the character and pitch of the murmur at the two areas are different, then there is justification for a diagnosis of two distinct murmurs (e.g. a high-pitched, blowing systolic murmur of mitral regurgitation associated with the harsh and low-pitched systolic murmur of aortic stenosis)

**6 Pitch.** The pitch of a murmur may be of value in the diagnosis of the murmur. Blowing murmurs (e.g. the systolic murmur of mitral regurgitation) are usually of a higher pitch than rumbling or rolling murmurs (e.g. the diastolic murmur of mitral stenosis). The pitch of a murmur, taken in conjunction with its other characteristics, frequently facilitates its recognition or identification

A murmur is made up of "random" vibrations of many frequencies. High frequency vibrations usually cause a high-pitched and low frequency vibrations, a low-pitched murmur. A high-pitched murmur, such as that of aortic regurgitation or small ventricular defect, usually suggests a high velocity of blood flow, a high pressure differential and a small orifice. A low-pitched murmur, on the other hand, such as that of mitral stenosis, suggests low velocity, small pressure difference and a large orifice.

Murmurs may be either high-pitched, medium-pitched or low-pitched. A high-pitched murmur (as in aortic regurgitation) is usually best heard either with the Bowles type of chest-piece or by the naked ear, a low-pitched murmur (as in mitral stenosis) on the other hand, is heard better with the bell type of chestpiece.

By varying the force or pressure of the chestpiece, the sensitivity for pitch may be altered to some extent. Whilst a high-pitched murmur, such as that of aortic regurgitation, requires a firm or forcible application of the diaphragmatic chest-piece, a low-pitched murmur such as that of mitral stenosis, is best heard with very light application of the bell chestpiece.

It has been demonstrated that a murmur varies directly with the velocity of blood flow across the area of murmur production. The velocity in turn is often closely related to the pressure head which drives the blood across the murmur generating area. For instance the velocity of flow across a small VSD gives rise to a loud murmur whereas a large flow at low velocity through an ASD produces no murmur. Also the frequency of a murmur bears a similar relationship to the velocity of blood flow, as does the intensity of a murmur. Low velocity with a small pressure head across a stenosed mitral valve produces low-pitched murmurs, while the large pressure difference across the aortic valve in aortic insufficiency causes high-pitched murmurs. Occasionally the frequency composition of the same systolic murmur may vary depending upon the area auscultated e.g. the ejection systolic murmur of aortic stenosis is higher pitched at the apex than at the base. (This supports the Vortex shedding theory, according to which the murmur heard at the site of production or downstream is lower pitched than upstream).

**7 Transmission or direction of selective propagation** The *propagation* or *transmission* of a murmur depends on the following factors (in order of importance) (a) The intensity or loudness of the murmur. The louder the murmur, the further it is transmitted. Thus, the loud murmur of aortic stenosis may be heard over the head or elbows. (b) The conducting nature of adjacent tissues. Whilst muscle and bone are good conductors of sound, fat, fluid and air tend to dampen sound conduction. (c) The direction of the blood stream producing the murmur. This factor is probably concerned in the upward conduction into the neck of the aortic stenotic murmur and the murmur of pulmonary stenosis (which is transmitted better on the left side of the neck), and the downward conduction of the aortic regurgitant murmur along the left sternal border due to the regurgitant stream of blood flowing into the left ventricle. (d) Other factors, e.g. the place of insertion of the papillary muscle. This has been shown to play a part in the conduction of the mitral regurgitation murmur into the axilla. The conduction has been artificially altered in site and propagation by transplanting the papillary muscle elsewhere.

## 8 Effects of posture, respiration, exercise and other manoeuvres

**POSTURAL EFFECTS** It is advisable to study all cardiovascular murmurs in both the recumbent and the upright (either standing or sitting) postures. Certain murmurs (e.g. the diastolic or presystolic murmur of mitral stenosis) are better heard or only heard during recumbency (or left lateral decubitus). Some murmurs (e.g. the diastolic murmur of aortic regurgitation) are better heard or only heard in the upright (or stooping forward) posture. Insignificant murmurs are markedly influenced by posture tending to disappear in the upright posture, a feature of some diagnostic value. In obstructive cardiomyopathy prompt squatting causes the systolic murmur to become faint or inaudible, whereas the systolic murmurs of mitral regurgitation and aortic stenosis tend to be louder.

Of the two commonly used postures (*viz.* recumbent and upright), the *upright posture* (either standing or sitting) is considered more dependable or satisfactory for the following reasons: (1) it tends to eliminate unimportant or insignificant murmurs, thus simplifying the task of diagnosis, (b) it tends to bring out otherwise inaudible aortic regurgitant murmurs, (c) it facilitates recognition of the mitral systolic murmur from other murmurs (such as murmur of VSD). It is our observation that during recumbency, the systolic mitral regurgitant murmur is frequently heard best in an anomalous position (e.g. in the fourth left interspace midway between the sternum and cardiac apex), a position in which it is likely to be mistaken for some other murmur. This can be prevented by adopting the upright posture, which shifts the mitral systolic murmur back to its normal apical position.

**EFFECT OF RESPIRATION** All murmurs should be auscultated during and at the height of both inspiration and expiration, as well as during holding of the breath after expiration. The great majority of organic murmurs become louder during expiration (the interposed lung tissue being less) and fainter during inspiration. In the case of cardiopulmonary murmurs, on the other hand, the murmur tends to disappear at the height of both inspiration and expiration and is maximal in mid-inspiration and mid-expiration. Some faint murmurs (such as the diastolic murmur of aortic regurgitation) may only be audible at the end of expiration. Innocent murmurs may be accentuated by inspiration as also the systolic and diastolic murmurs of tricuspid regurgitation and stenosis (Carvalho's sign). Accentuation or increased audibility of a tricuspid murmur is most obvious during slow and deep inspiration following on a full expiration. This sign is however negative, if deep inspiration (or apnoea) is maintained for any length of time. Accentuation of the *diastolic* murmur of *tricuspid stenosis* during inspiration has been attributed to markedly increased velocity and quantity of blood (increase of over 30 per cent) through the tricuspid orifice (even in advanced cases of valve stenosis) during this phase of respiration. Because of lowering of intrathoracic pressure during inspiration, there is a rise in the "peripheral venous—right ventricular

filling" gradient (or the right atrioventricular transvalvular diastolic gradient), which in turn increases and accelerates blood flow across the valve-barrier. In the case of the systolic murmur of *tricuspid regurgitation*, accentuation of the murmur during inspiration is due to (a) increased rate of regurgitation because of the increased stroke volume of the right ventricle during systole, and (b) increased traction of the chordae tendinae on the valve leaflets (with increase in size of the tricuspid aperture and increased reflux of blood), because of increase in the volume of the right ventricular chamber.

**EFFECT OF EXERCISE.** Exercise, by increasing the rate of flow of blood, may help to bring out murmurs which are otherwise faint or inaudible. This is particularly true for (1) the diastolic murmur of mitral stenosis and (2) the so-called innocent or insignificant murmur (in the pulmonary or mitral area).

Exercise may cause the appearance of a transitory or evanescent systolic murmur in normal healthy individuals, particularly in children and adolescents. This factor must therefore be taken into account before regarding a systolic murmur as significant.

**VALSALVA MANOEUVRE.** Forced expiration against a closed glottis causes initially a sharp rise in blood pressure (phase I). As the straining continues, there follows a decrease in stroke volume, heart size, and mean arterial pressure (phase II). Innocent murmurs are hardly affected by this technique. During phase II the murmurs of aortic and pulmonary stenosis become fainter, on the other hand the murmur of HOCM intensifies due to reduction in left ventricular volume. In the case of systolic click and late systolic murmur syndrome, the murmur starts earlier and may become more musical. The manoeuvre also helps in differentiation of coronary artery fistula from extracardiac shunts. With Valsalva there is comparatively more reduction in systolic and pulse pressures than the pressures in the pulmonary circulation thereby reducing the pressure across the intracardiac fistula. Extracardiac shunts are not similarly affected.

**ISOMETRIC HAND GRIP.** Sustained isometric hand grip causes an increase in pulse rate and blood pressure. (Forceful clenching of the fist can serve a similar purpose). The effect on murmurs is: (a) Regurgitant (both aortic and mitral) become louder due to rise of aortic diastolic and ventricular systolic pressures respectively. (b) Diastolic murmur of mitral stenosis becomes louder due to increased flow through mitral valve. (c) Murmur of valvar aortic stenosis and HOCM decreases. Sustained hand grip is contraindicated in presence of recent myocardial infarction, hypertension or cerebrovascular disease.

**OTHER MANOEUVRES.** (a) Occlusion of the internal jugular vein by light pressure over the vein, rotation of the neck (or Valsalva manoeuvre) makes a venous hum become faint or disappear. (b) External compression of the chest wall can accentuate the basal systolic murmur of straight back syndrome.

(c) Supraclavicular arterial bruit can be silenced or muffled by marked extension of the shoulder

9 Effect of drugs and pharmacological agents. Some drugs by producing haemodynamic changes produce alteration of murmurs and heart sounds

(a) PRESSOR AGENTS like phenylephrine These produce rise in systemic arterial pressure causing (i) Intensification of left-sided regurgitant murmurs (ii) In Fallot's tetralogy the systolic murmur is similarly intensified (iii) A small VSD murmur becomes louder due to rise in left-to-right gradient (iv) The diastolic component of PDA murmur may become obvious due to the same mechanism (v) In HOCM the systolic murmur may become fainter or inaudible

(b) AMYL NITRITE Inhalation of amyl nitrite causes sudden peripheral vasodilatation, a rapid fall in systemic blood pressure, increased venous return and cardiac output Because of ease of administration and safety it can be used for diagnosis of (i) Left sided regurgitant murmurs, VSD, PDA, and murmurs of systemic A-V fistulae become shorter and softer (ii) Right sided regurgitant murmurs (TR) and all ejection murmurs become louder due to increased venous return and increased forward flow (provided there is no heart failure), with the exception of the ejection murmur in tetralogy of Fallot which softens because blood is diverted down the aorta instead of across the pulmonary stenosis (iii) It can help to distinguish Austin Flint murmur from a mitral stenosis murmur The Austin Flint murmur fades as the aortic regurgitation decreases whilst the murmur of MS is accentuated (iv) The murmur of systolic click, late-systolic murmur syndrome increases in duration (v) In acute papillary muscle dysfunction the late systolic murmur becomes pansystolic and louder

10 Presence of thrill. A fair percentage of murmurs are accompanied by thrills Thrills are usually associated with low frequency or harsh murmurs and therefore commonly associated with stenotic or obstructive lesions Thrills are usually absent with high-pitched or high frequency blowing murmurs (e.g. murmur of aortic regurgitation)

The presence of a thrill, in conjunction with a murmur, at the apex, usually suggests mitral stenosis, in the aortic area, either aortic stenosis or aneurysm, and in the pulmonary area, some form of congenital heart disease

## TYPES OF MURMURS

It is convenient to classify cardiovascular murmurs into the following main varieties

1 *Innocent* (normal, benign or insignificant) *murmurs*, inclusive of (a) the musical or vibratory systolic murmur, (b) the supraclavicular arterial bruit, (c) the pulmonary ejection murmur, (d) the cardiorespiratory murmur, (e) the mammary souffle, and (f) the venous hum



2 *Physiological (or hyperkinetic) murmurs*, secondary to hyperdynamic circulatory states (or accelerated circulation), as in case of severe anaemias, fevers, thyrotoxicosis and beri-beri heart disease

3 *Functional or relative murmurs* (a) *Dilatational murmurs* secondary to dilatation of cardiac chambers or blood vessels, but not associated with any structural abnormality such as valvular lesions, narrowing of vessels or shunts. The systolic apical murmur of hypertensive cardiac enlargement, usually ascribed to non-valvular, relative or functional mitral insufficiency (secondary to A-V ring-dilatation) belongs to this category. (b) *Flow murmurs*. In V S D and P D A there may be a diastolic murmur from increased blood flow through the mitral valve. With a large A S D a diastolic murmur may be heard at the apex due to increased flow through the tricuspid valve. The term haemic murmur suggests a murmur associated with hyperkinetic circulation. In majority of such cases the murmur is due to increased pulmonary blood flow.

4 *Organic murmurs*. Structural disease or deformity of the heart, usually of the nature of valvular stenosis or insufficiency, narrowing of a blood vessel (e.g. infundibular aortic stenosis) or arteriovenous shunt, is responsible for this variety of murmur.

#### CLASSIFICATION OF MURMURS

A murmur may be heard in systole, or in diastole, or continuously throughout both systole and diastole.

Since the time of Laennec, numerous classifications of heart murmurs have been suggested and later rejected. For practical purposes, the following classification, based on the timing of murmurs in relation to the first and second heart sounds, appears the most convenient.

##### Systolic Murmurs

##### I *Midsystolic, ejection or forward flow murmurs*

(a) Aortic	(i) Obstructive	Supravalvar Valvar
	(ii) Increased flow	Infravalvar Aortic regurgitation Complete heart block
	(iii) Aortic sclerosis	
(b) Pulmonary	(i) Obstructive	Supravalvar Valvar
	(ii) Increased flow	Infravalvar Physiological
	(iii) Dilatation of distal chamber	Pulmonary hypertension
(c) Innocent murmur (Early, mid or late systolic)		

II *Pansystolic, regurgitant or back flow murmurs*

(a) A-V valve incompetence

Mitral regurgitation  
Tricuspid regurgitation

(b) Left to right shunt at  
ventricular level or beyond

V S D  
P D A (early)

III *Late systolic murmurs*

Coarctation of aorta  
Hypertrophic obstructive cardiomyopathy  
Disease of mitral chordae or papillary muscle

IMPORTANT CARDIOVASCULAR MURMURS

Certain murmurs are of more clinical significance than others, both from the point of view of diagnosis and of prognosis. An elucidation of the numerous characteristics of a murmur frequently permits an insight into the nature of the causative lesion, as well as its subsequent behaviour or outlook. Of the large number of murmurs known, the following are singled out for special consideration in view of their importance, prevalence or clinical significance.

INNOCENT MURMURS

The terms "functional murmur" and "normal murmur", frequently employed for this variety of cardiovascular murmur, appear undesirable, being scientifically inaccurate and are therefore best avoided. The term "functional murmur" has been widely used in the past to include a variety of different conditions, both significant and insignificant. Whilst all murmurs of non-valvular origin (including murmurs secondary to dilatation of cardiac chambers) have been classed as "functional" by some, others have restricted the use of this term to murmurs unassociated with structural disease or disorder of the heart, and including both innocent and hyperkinetic murmurs. In normal subjects, particularly in children and during exercise, emotion or fever, it is common to hear soft and "usually" short, systolic or continuous, so-called "innocent" murmurs. Usually heard best over the apical and pulmonary areas, innocent murmurs may be loudest over the neck region or along the left sternal border. Accentuated by exercise and change of posture, these murmurs tend to disappear after childhood, are perfectly harmless and associated with normal hearts.

As claimed by Potain, many years ago, there is no simple method of distinguishing an "innocent" from a clinically "significant" murmur, the differentiation resting mainly on a careful appraisal of the cardiovascular system in general and of the various characteristics of the murmur (in question) in particular. The great majority of innocent murmurs are systolic.

**CHARACTERISTICS OF INNOCENT MURMURS** This depends mainly on one's familiarity with the clinical characteristics of such murmurs. The following features about innocent murmurs are worthy of note in this regard, being capable of differentiating such murmurs from the clinically significant ones

(1) *Timing and duration* With the exception of the venous hum and mammary souffle (continuous murmurs, which can be obliterated by compression of superficial vessels in the area of murmur), most (if not all) innocent murmurs are systolic in timing. Innocent murmurs are never pansystolic (as in case of mitral or tricuspid insufficiency and ventricular septal defects) being usually of ejection type. Whilst a pansystolic murmur is always significant, an ejection type of systolic murmur may be innocent or harmful.

Although diastolic murmurs may be encountered occasionally in cases of severe hypertension (because of dilatation of aorta), thyrotoxicosis, severe anaemias and congenital shunt-defects, in the absence of valvular heart disease, they cannot be regarded as "innocent", being associated with clinically significant diseases. It is important to note that whilst pansystolic murmurs are always significant, early, mid and late systolic murmurs may be significant or innocent, the ultimate decision to be based on associated manifestations.

(2) *Intensity* Whilst the majority of innocent murmurs are "soft" or "weak" (Grade I to III), the majority of significant murmurs are loud in intensity, there being exceptions in each group. Thus, the clinically significant diastolic murmur of aortic insufficiency may be almost inaudible, whilst an innocent musical murmur at the apex may be easy of detection.

(3) *Location* The site of maximum localization of a murmur may or may not be of diagnostic value. Whilst murmurs best heard in the apical area may be organic or innocent and those maximal in the pulmonary area frequently innocent, murmurs heard best in the aortic area or second right interspace are nearly always organic.

(4) *Radiation* Wide propagation or transmission of a murmur is more in favour of an organic than innocent murmur, although exceptions to this rule are common enough. Since transmission of a murmur depends more or less on its intensity, it is but natural that the usually loud organic murmur should be more widely propagated than the usually soft innocent one.

(5) *Quality* Innocent murmurs (unlike organic or significant ones) are usually of uniform pitch and frequency and frequently display (particularly at the apex) a characteristic musical, twanging-string, groaning or vibratory quality. Whilst a "blowing" type of murmur may be innocent or significant, a rumbling, rasping or grating type of murmur at the apex should suggest organic heart disease.

(6) *Effect of posture* Innocent murmurs, are, as a rule, markedly affected by change of posture, respiration and exercise. Frequently loud or well heard

during recumbency, they tend to become faint or disappear in the sitting up or erect posture. The effect of respiration on innocent murmurs is somewhat variable, although the majority of them become fainter during inspiration. Disappearance of a loud murmur on inspiration is highly suggestive of an innocent murmur, although exceptions to this rule are common enough. An occasional innocent murmur may become faint during expiration, whilst a faint organic one may show attenuation during inspiration. The effect of exercise on innocent murmurs is variable (both accentuation and attenuation having been reported after exercise). The effect of exercise on the intensity of a murmur is therefore of little or no diagnostic significance.

(7) *Second heart sound* The second heart sound shows normal respiratory variations of wider splitting on inspiration and narrowing of the splitting on expiration.

(8) *Age* The age incidence may or may not be of value in differential diagnosis. Whilst in children and young adults, both innocent and clinically significant murmurs are common enough, in individuals over the age of 40 or 50 years, organic murmurs are more or less the rule. Innocent murmurs are commonly encountered during pregnancy, fevers and after exercise.

(9) *Absence of evidence of heart disease* Whilst a total absence of all associated cardiovascular abnormalities or features, suggestive of organic involvement (such as a positive history of rheumatic fever or syphilis in the past), symptoms and signs of heart disease (such as dyspnoea, cyanosis, clubbing, oedema and cardiac enlargement), evidence of congestive heart failure, and instrumental signs of cardiac involvement, favours the diagnosis of an "innocent" rather than "organic" murmur, the presence of such features does not necessarily rule out the existence of a superimposed innocent murmur.

In the final assessment of a murmur, attention to the following features is essential: *timing* of the murmur (diastolic and pansystolic murmurs being always organic), its intensity (loud murmurs are usually organic), presence or absence of signs of cardiac enlargement, heart disease or failure of the heart, and normal or abnormal findings during fluoroscopy, radiography and electrocardiography.

### **Types of innocent murmurs**

(1) **THE MUSICAL OR VIBRATORY MURMUR** (Still's murmur) is the commonest innocent precordial murmur. Usually confined to childhood, this musical low-pitched murmur, with its peculiar "groaning" or "twanging string" character and uniform frequency, is usually heard best at the apex, although it may be audible also along the left sternal margin and pulmonary area. Best heard in the supine position, the musical murmur is usually heard best with the bell of the stethoscope, is mid-systolic or early systolic in timing, and is associated with normal heart sounds. The third heart sound is frequently audible. The mode of production of this murmur remains obscure.

Differential diagnosis is from the systolic murmurs of mitral insufficiency and ventricular septal defect. In favour of its being innocent rather than organic are the following features, viz a regular or uniform frequency, short duration, mid-systolic timing, absence of thrill, normal splitting of the second sound, absence of other murmurs (e.g. diastolic), lack of transmission or radiation and moderate intensity. A musical murmur is never pansystolic.

(2) THE PULMONARY EJECTION MURMUR. Common in children, young adults with thin chest-walls and pregnant women, this relatively soft and short murmur is one of the most frequently observed varieties of innocent murmur. Heard best in the pulmonary area, mid systolic in timing and usually unassociated with a thrill, the pulmonary ejection murmur tends to become *faint* (or disappears) in the upright posture and during inspiration. It is usually *accentuated* by exercise and accelerated circulation (as in hyperdynamic circulatory states).

Ascribed to a rapid ejection of blood into the pulmonary artery during systole, this innocent murmur must be distinguished from the somewhat similar pulmonary ejection murmurs of atrial septal defect and pulmonary valve stenosis.

(3) THE SUPRACLAVICULAR ARTERIAL BRUIT (Carotid and cervical arterial bruits). Best heard over the supraclavicular fossa and carotid vessels and extending (at times) down to the aortic or pulmonary area, this short, early systolic fairly intense (Grade 2 to 4), low-frequency, ejection type of murmur is quite common in normal healthy children and young adults and is frequently heard best in the right supraclavicular fossa. The bruit may be particularly loud in patients with hyperdynamic circulatory states because of increased velocity of blood flow. It can often be obliterated by compression of the subclavian artery and hyperextension of the shoulders.

The mode of production and site of origin of the murmur, have not as yet been established, although "turbulence" at the bifurcation of the innominate artery into the subclavian and right common carotid has been regarded as causative by some authorities. Since compression of the carotid and subclavian arteries does not always obliterate this bruit, the latter does not always arise from the distal parts of these vessels. In such cases, possibility of the murmur arising more proximally (say in the innominate artery or aorta) cannot be ruled out.

When the supraclavicular arterial bruit is transmitted to below the clavicle (to the aortic or pulmonary area), its differential diagnosis from the murmurs of aortic stenosis, pulmonary stenosis and atrial septal defect may prove difficult, unless it is remembered that the supraclavicular bruit (unlike the other murmurs) is always maximal over the supraclavicular fossa or neck vessels. The murmurs of atherosclerosis in elderly subjects, although often heard best over the neck vessels also, are as a rule louder and longer in duration, of higher

pitch, at times continuous, and associated with other manifestations of atherosclerosis

(4) **THE CARDIORESPIRATORY (or cardiopulmonary) MURMUR** An extracardiac murmur usually due to the sucking-in or squeezing action of the heart on adjacent lung tissue (resulting in aspiration or pushing out of air from the lung), the cardiorespiratory murmur may at times be due to vibrations of pericardial or pleural layers or adhesions. Bizarre and variable of nature, described by some as a "systolic whoop" and compared by Laennec to the sound of "a soft wooden file or saw", the cardiorespiratory murmur is best heard at the apex, sounds more superficial than an intracardiac murmur, is almost invariably systolic or late systolic (rarely diastolic) and is frequently initiated by a loud systolic click. It tends to disappear during deep inspiration, complete expiration and holding of the breath, and is loudest in mid-inspiration or mid-expiration (with the lungs partially inflated), features of diagnostic value.

Although rightly regarded as innocent, the cardiorespiratory murmur and systolic click may at times be associated with organic heart disease, pericarditis, pleurisy, ascites, pectus excavatum, Marfan's syndrome or pregnancy.

#### (5) OTHER BENIGN MURMURS

(a) *Aortic valve sclerosis* The aortic ejection murmur of middle and old age, although not innocent, can be considered benign. It is less loud and less harsh than the murmur of aortic stenosis and shows little upward radiation. It however tends to be transmitted to the apex, where it may be louder than at the base. A thrill is rare. The diagnosis of this harmless murmur is suggested by the age of the patient, the presence of hypertension or of aortic dilatation, and the absence of other valvular lesions.

(b) *Pectus excavatum* (Funnel chest) The effects on the heart depend on the degree of backward bulge of the sternum. A mid-systolic murmur soft to moderately loud is usually best heard along the left sternal edge. It probably results from the impact of the heart against the bulge of the sternum.

(c) *Straight-back syndrome* Absence of the normal anterior dorsal concavity reduces the antero-posterior diameter of the chest as in pectus excavatum. Thus the chest is compressed and there is kinking of the great vessels, and distortion of the outflow tract particularly of the right ventricle. A parasternal systolic murmur is not uncommon and the second heart sound tends to be widely split. The murmur decreases on deep inspiration and may become louder on compressing the chest externally.

(d) *Severe kyphoscoliosis* An ejection systolic murmur localised to the pulmonary area encountered in gross deformities of the spine and thoracic cage either congenital or acquired. The murmur may be late systolic, soft to moderately loud, decreases in intensity on inspiration and in the sitting posture.

- |                     |                                |
|---------------------|--------------------------------|
| (6) VENOUS HUM      | } See under continuous murmurs |
| (7) MAMMARY SOUFFLE |                                |

## SIGNIFICANT MURMURS

## Systolic Murmurs

The classification of systolic murmurs into either midsystolic ejection or pansystolic regurgitant murmurs has been almost universally adopted. Although there are bound to be deficiencies in any single classification, this categorization has become popular because it has a physiological as well as descriptive basis. For completeness, variations of ejection and regurgitant murmurs will also be discussed.

## EJECTION SYSTOLIC MURMURS

A MIDSYSTOLIC EJECTION MURMUR is caused by forward flow through either the aortic or pulmonary valve or outflow tract. It may be seen in the following haemodynamic conditions: (a) Obstruction of the right or left ventricular outflow tract, (b) increased rate of ejection through the normal valve, (c) valvular damage without stenosis, (d) dilatation of the great vessel beyond the valve, or (e) any combination of these factors. The murmur begins when the pressure in the respective ventricle exceeds the aortic or pulmonary artery pressure sufficiently to open the aortic or pulmonary valve. As a result, there is a delay between the first heart sound which occurs immediately after atrioventricular closure and the beginning of the murmur. The murmur then waxes and wanes in a crescendo-decrescendo fashion which is described as 'diamond shaped' in configuration (Fig 9.21). The murmur invariably stops before the semilunar valve closure of the respective ventricle is heard.

The contour of the time intensity pattern or 'envelope' of a murmur corresponds to the contour of the flow pulse passing through the region at the time of production. Thus, not only the overall intensity of the murmur is proportional to the mean rate of left ventricular ejection, but the shape of the murmur will depend upon the instantaneous flow characteristics during the period of ejection. During normal left ventricular ejection, a disproportionately large volume flow occurs in early systole. If velocity of the flow exceeds the murmur threshold, a short ejection murmur results and its envelope corresponds to the flow pattern. If the stroke volume of the ventricle is increased, this pattern of ejection persists in an exaggerated fashion, the resultant murmur has a tendency to peak early in systole and fade out about midway through the ejection phase. These murmurs have been called 'kite shaped' and are commonly found in high output states or conditions such as aortic regurgitation or complete heart block where the stroke volume is high. It should be remembered that the flow characteristics of normal right ventricular ejection are quite different. Early ejection rates are not early as high with the flow curve peaking somewhat later and having a more rounded contour. This flow pattern serves to explain some of the long systolic ejection murmurs heard in atrial

septal defects and the 'straight back syndrome' where no gradient can be found across the right ventricular outflow tract

With true valvular obstruction rapid early ejection is not possible, the aortic flow pattern becomes rounded resulting in more symmetrical murmur of aortic stenosis. If obstruction is severe systole is prolonged. However, the murmur always stops before the closure sound of the respective ventricle, although it may envelope the closure sound of the opposite side of the circulation. Because of the high correlation between the shape of the murmur and its underlying flow characteristics, careful attention during auscultation must be paid to the shape and duration of the murmur as well as its intensity. It should be noted that systolic ejection murmurs are very sensitive to various physiologic and pharmacologic manoeuvres which alter cardiac output, their response may readily differentiate them from pansystolic regurgitant murmurs.

### A Aortic systolic murmurs

1 *Left ventricular outflow tract murmurs* Left ventricular outflow tract obstruction may be congenital or acquired, and it may be localised to the valvular, supravulvar, or subvalvular level. Subvalvular stenosis may be caused by either a discrete fibrous band or by obstructing muscle (hypertrophic subaortic stenosis). The table below gives a summary of the differential diagnosis of various forms of left ventricular outflow tract obstruction.

Clinical features	Congenital A S			Acquired A S	HOCM
	Valvar	Sub-valvar	Supra-valvar		
Physical appearance	Normal	Normal	Elfin facies	Normal	Normal
Arterial pulse	Slow rise and sustained peak		*Right brachial & carotid more than left	Slow rise, sustained peak	Brisk rise, unsustained double peak
Apical impulse	Sustained and single				Sustained, may be double
Aortic ejection sound	Typical	Rare	Rare	Uncommon	Very rare
Maximum intensity of ejection murmur	First or second right interspace		First right interspace & over right carotid	First or second right interspace	Apex, lower left sternal edge
Splitting of 2nd sound	Usually normal or single			Usually single or paradoxical	Usually paradoxical or single
Intensity of A <sub>2</sub>	Normal or decreased			Decreased or absent	Normal or decreased
Murmur of A R	Common	Common	Uncommon	Common	Very rare

#### Differential Diagnosis of left ventricular outflow obstruction

\* This may relate to the tendency of a jet stream to adhere to a vessel wall (Coanda effect), and selective streaming of blood into the innominate artery.



The following general statements can be made. The intensity of the aortic ejection and closure sounds correlates primarily with valve mobility, and no relationship exists between its presence and the severity of the stenotic lesion. However its presence with rare exceptions suggests obstruction at the valvular level. With significant calcification of the valve, both the ejection sound and the aortic closure may be markedly decreased or absent. With increasing severity of stenosis, the gradient increases and the duration of ejection increases if flow is maintained. As a result, the murmur becomes louder and longer with a tendency towards later peaking. With increasing severity of stenosis the duration of left ventricular ejection is prolonged selectively and produce narrow, single or paradoxical splitting. All haemodynamically significant fixed orifice obstructions have slow rising anacrotic pulses. Any physiologic or pharmacologic manoeuvre which increases flow will increase the intensity of the murmur. Similarly, it should be remembered that occasionally "silent" aortic stenosis can be present in the older age group when cardiac output is markedly decreased. Silent aortic stenosis should also be kept in mind when a patient presents with severe congestive failure and no obvious underlying aetiology.

The murmur of significant fixed left ventricular outflow tract obstruction is usually heard best in the second right and the second and third left intercostal spaces. Occasionally the murmur becomes softer as one goes down from the aortic to the mitral area (inching), and then again increases in intensity (hour-glass conduction). With radiation to the apex, the high frequency components of the murmur predominate, and the apical murmur has a high pitch and often a musical component. Such change in the pitch of the murmur may prompt the examiner to call the apical murmur a separate murmur. Beat-to-beat variation in the intensity of the murmur of aortic stenosis have been noted in *pulsus alternans* and *A-V dissociation*.

Normally, aortic valve closure precedes pulmonary valve closure, in severe aortic stenosis or left bundle branch block, however, the left ventricular systole may be sufficiently prolonged to reverse the order of valve closure, the pulmonary valve closing before the aortic. In such a case, the murmur of aortic stenosis may cause some confusion by drowning the pulmonary component of the second sound, but even then it always stops short of the aortic component and the clear interval between the two events clinches the diagnosis, provided the aortic component is not too soft or absent (as in cases of severe stenosis).

The murmur of aortic stenosis, although classically loud, widely transmitted and accompanied by a thrill, is at times soft, poorly transmitted or unaccompanied by a thrill, as in the case of severe left ventricular failure (where the rate of flow of blood from the ventricle is impaired), associated mitral regurgitation (where part of the blood from the left ventricle is diverted back to the left auricle) or emphysema (due to defective conduction of murmur to the chest wall). The murmur of aortic stenosis may be best heard at the apex (as in some cases of emphysema where conduction of the murmur to the basal areas is poor), in such a case, the interval between the cessation of the murmur and the second sound, and the slowing or anacrotic nature of the pulse, give away the correct diagnosis.

The later the peak or *crescendo* of the murmur in systole, the greater is usually the degree of stenosis. In the case of an aortic ejection murmur, the peak may occur early enough to be

drowned by a loud systolic ejection click, in which case, the murmur may sound decrescendo just after the click. The typical crescendo-decrescendo murmur of aortic stenosis is graphically described as "diamond-shaped" or as having a "Christmas tree configuration".

In calcific aortic stenosis the murmur, although loud and noisy in the aortic area (due to the "jet" or flow into the aorta), may sound *musical* along the left sternal border or in the apicosternal area (due to musical vibration of the stenotic valve diaphragm), the characteristic difference in the quality of the murmur, at the two sites, being referred to as the Gallavardin phenomenon.

**2 Coarctation of aorta** The murmur of coarctation is closely similar to that of aortic stenosis, the crescendo being somewhat delayed, because of the more distal site of narrowing of the blood vessel. It is a loud murmur, best heard either at the base of the heart or at the back (in the interscapular region), frequently accompanied by a thrill and associated with other diagnostic signs (such as brachial hypertension, weak or absent femoral pulses, visible and palpable collateral vessels over the thoracic wall, and additional murmurs and thrills over the dilated vessels).

**3 Functional murmurs** Such murmurs are typically of short duration and have a kite-shaped configuration. (a) Flow murmurs. They may be introduced by an aortic ejection sound (of the root type) which is not as loud as that associated with valvular aortic stenosis. These murmurs are rarely associated with a thrill, although in severe aortic regurgitation a thrill may be present in association with the systolic ejection murmur. Frequently in older people, these murmurs radiate to the apex. These murmurs are not associated with abnormal carotid upstrokes. (b) Dilatational murmurs. Functional aortic systolic murmurs may be encountered in syphilitic aortitis and aortic aneurysm due to dilatation of the ascending aorta.

## **B Pulmonary systolic murmurs**

**1 RIGHT VENTRICULAR OUTFLOW TRACT OBSTRUCTION** Right ventricular outflow tract obstructions are usually congenital and may be at the level of the valve, infundibulum, or pulmonary branches. Isolated infundibular stenosis with an intact septum is rare and is usually associated with a large ventricular septal defect (tetralogy of Fallot).

Valvular and infundibular pulmonary stenosis differ in the following ways. In isolated infundibular obstruction, a pulmonary ejection sound is never heard, and pulmonary closure sound is not audible except in the mildest cases. In more severe cases the width of splitting is significantly greater than in valvular stenosis of comparable severity. The site of maximum intensity of the murmur is of little help in differentiating the site of obstruction. Pure infundibular stenosis with an intact ventricular septum may be differentiated from tetralogy of Fallot by noting the marked increase in intensity of the ejection murmur after inhalation of amyl nitrite, the murmur of tetralogy shortens and decreases in intensity.

The murmur of valvular or infundibular pulmonary stenosis is closely similar to that of aortic stenosis, being usually a loud, harsh or musical, mid-systolic murmur with an early crescendo and ending clearly before the pulmonary component of the second sound. In severe pulmonary stenosis, because of prolonged right ventricular systole, pulmonary valve closure may be markedly delayed, in such a case, the murmur may be long enough to mask or cover the earlier aortic component but never the delayed pulmonary component which is softly audible in most cases.

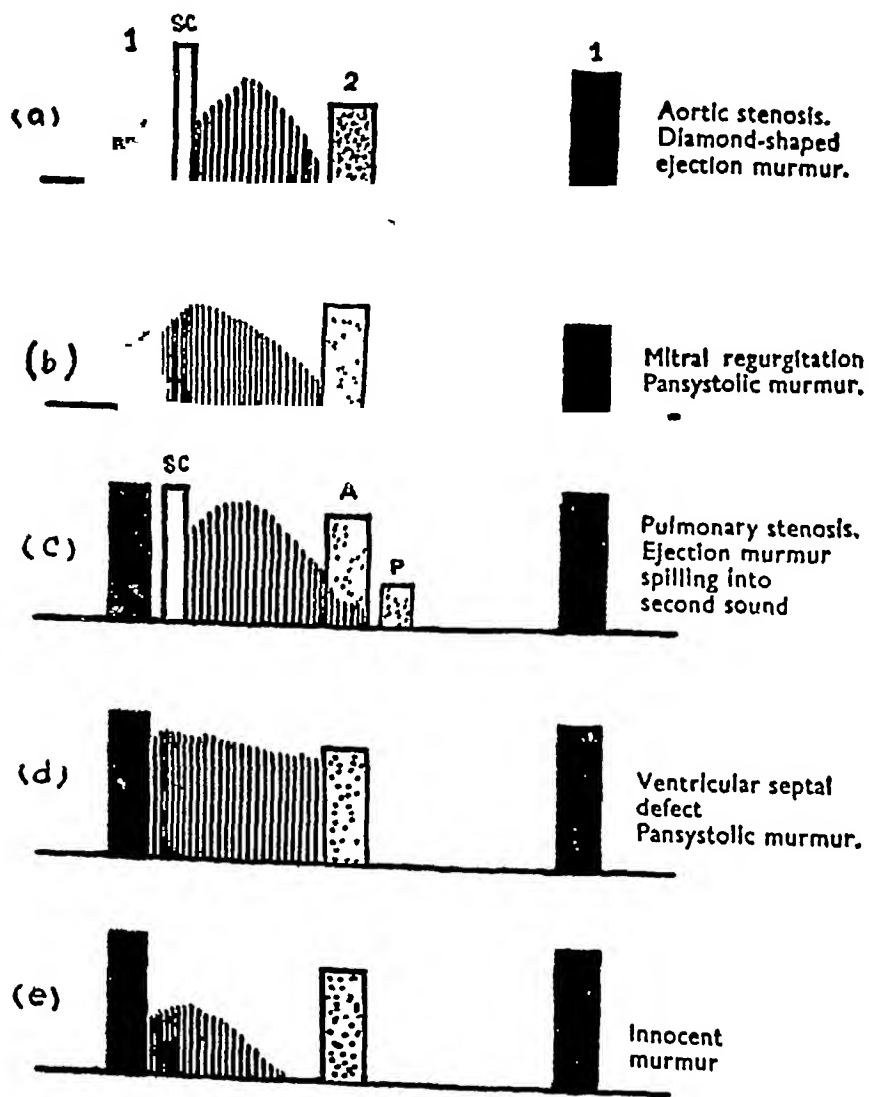


Fig 9 21 Systolic cardiac murmurs

2 **TETRALOGY OF FALLOT** In Fallot's tetralogy, pulmonary stenosis is associated with a large ventricular septal defect. Much of the blood from the right ventricle is diverted to the dextroposed aorta and the pulmonary artery flow being correspondingly lessened, the systolic murmur (due to pulmonary stenosis) is frequently softer and shorter than in isolated pulmonary stenosis and the pulmonary component absent, making the classical nature of the murmur difficult to appreciate. The right to left shunt, through the wide ventricular septal defect in such cases, is not as a rule severe enough to initiate a murmur. The more severe the obstruction, the less blood flow there is across the pulmonary outflow tract and more blood is shunted through the septal defect, with increased severity of pulmonary infundibular obstruction, the murmur shortens and the cyanosis deepens. During the hyperdynamic episodes which children with tetralogy of Fallot may experience, the murmur may actually disappear. This suggests that such spells can be attributed to infundibular spasm with complete obstruction of the pulmonary outflow tract.

In the severest forms of tetralogy and pulmonary atresia (frequently called pseudo-truncus arteriosus), there is no flow across the pulmonary valve and no pulmonary ejection murmur. Instead, one hears a continuous murmur of increased flow through the enlarged bronchial arteries, and invariably a loud ejection sound is present.

In *branch stenosis of the pulmonary artery*, there is a systolic ejection murmur of varying intensity at the upper left sternal border which is widely transmitted to the right chest, back, and to both axillae. The murmur is usually less harsh and of a higher pitch than the murmur of valvular stenosis and is accompanied by a thrill. With more peripheral stenosis, systolic ejection murmurs or even continuous murmurs may be heard over the lung fields.

3 **ATRIAL SEPTAL DEFECT** In case of atrial septal defect and in anomalous pulmonary venous return, there is a left to right shunt, the right ventricle receives more blood, its stroke volume is therefore increased with increased flow of blood into the pulmonary artery, resulting in a moderately loud systolic ejection murmur. The peak of the murmur is usually early, because of the increased rate of ejection early in systole. If a large left-to-right shunt is present, a tricuspid diastolic flow rumble may be heard. In the presence of high pulmonary vascular resistance with diminution and a reversal of the shunt, the tricuspid rumble will disappear, the pulmonary ejection murmur will shorten, and the wide splitting of the second sound tends to disappear. A diastolic murmur of pulmonary incompetence commonly develops.

4 **FUNCTIONAL MURMURS** (a) *Increased cardiac output* In states of anaemia, thyrotoxicosis, peripheral A-V fistulae, pregnancy, fever and exercise, and with slow heart rates (as in complete heart block), the increased cardiac output is not selective or confined to the right ventricle (as in atrial septal defect) but affects both ventricles. The increased stroke volume results in an early systolic ejection murmur. In such cases, both ventricles have high stroke

outputs, the aortic and pulmonary valve closure sounds bear a normal relationship to each other, being fused in expiration and separate in inspiration thus distinguishing the condition from that of atrial septal defect

Murmurs associated with hyperkinetic or high output states such as exercise or fever are best heard at the base because the velocity of blood flow is most rapid at the origin of aorta and pulmonary artery. The reason why these murmurs are more common in the pulmonary area is probably due to the following reasons: (i) the membranous portion of the septum bulges to the right and the outflow tract of the right ventricle has a crescentic shape, (ii) the conus of the right ventricle is encircled by myocardial fibres which when they contract in systole reduce the cross-sectional area, (iii) and lastly because the pulmonary valve cannot open fully and gives rise to a triangular orifice

The ejection murmur of high pulmonary artery flow may display the superficial scratchy quality of a pericardial friction rub. This is particularly so with thyrotoxicosis. The scratchy superficiality may be due to the anatomical nearness of the dilated pulmonary artery in these cases.

(b) *Pulmonary artery dilatation* An important cause of pulmonary ejection murmur is a dilatation of the pulmonary artery, usually from pulmonary hypertension or may be an isolated anomaly. In such cases the murmur may be soft or loud, is usually preceded by an ejection click or sound, and shows an early peak. The peak may be early enough to be masked by the ejection click, giving an apparently decrescendo character to the murmur. In addition, pulmonary murmurs of this type are heard in children and young adults, especially among women of asthenic habitus. These latter innocent murmurs are short and have a vibratory quality (hence called grunting or zipping murmurs).

A benign systolic ejection murmur is common in the straight back syndrome. This murmur may be associated with a tendency towards wide splitting of the second sound and a pulmonary ejection sound which varies with respiration, splitting may be relatively fixed.

## REGURGITANT SYSTOLIC MURMURS

### A. Pansystolic regurgitant murmurs

Usually associated with backward flow of blood (a) from ventricle to atrium through an incompetent mitral or tricuspid valve, or (b) left to right shunt through a ventricular septal defect or patent ductus arteriosus. Since there is a high pressure differential between the two chambers throughout systole, the murmurs are holosystolic in duration continuing right upto the second heart sound, high-pitched, blowing in quality, and plateau-like in configuration.

1 **MITRAL REGURGITATION** The most important single sign of mitral regurgitation is a pansystolic murmur (Fig 9.21), maximal over the apex or near it, usually loud and blowing, transmitted to the axilla or inferior angle of the left scapula, fainter during inspiration, occasionally associated with a thrill, and best heard with the Bowles type of chestpiece, because of high frequency vibrations. The pansystolic character of the murmur is due to

the fact that throughout entire systole the pumping chamber or left ventricle shows a much higher pressure than the receiving chamber or left atrium, even at the time of closure of the aortic and pulmonary valves. This may cause the murmur to obscure the second sound at the apex (by drowning the aortic component) but not at the pulmonary area, a fact which may be of value in differentiating a transmitted aortic from a true mitral murmur.

In mitral insufficiency, the output of the left ventricle is the sum of the forward plus the regurgitant systolic flow. Since in a steady state the diastolic flow has to be equal to the stroke output, there is a large atrioventricular inflow during each diastole. This increase in the rate of diastolic inflow during early diastole gives rise to a rapid ventricular filling sound. When the degree of regurgitation is severe, this sound often introduces a short diastolic rumble indicative of the high rate of filling without significant stenosis.

In chronic mitral insufficiency with a dilated left atrium, an atrial sound is not heard. In acute mitral insufficiency occurring on a previously normal valve, an atrial sound is common. This sound results from the forceful contraction of a normal atrium ejecting into the distended but normally compliant left ventricle.

**2 TRICUSPID REGURGITATION** The physical signs of tricuspid valve disease are frequently obscured or complicated by those of the underlying or primary condition, e.g. mitral stenosis or emphysema.

The systolic murmur of tricuspid regurgitation is closely similar to that of mitral regurgitation (Fig. 9.21). It is a long, pansystolic murmur, usually blowing and high-pitched, best heard over the lower end of the sternum or inner end of the fourth left interspace (sometimes heard equally well at the apex), variable in intensity but usually maximal during inspiration, with or without a thrill and usually associated with gross engorgement and systolic pulsation of the liver and neck veins.

Inspiratory accentuation of the murmur of tricuspid regurgitation is due to the increased filling of the right ventricle during inspiration and serves to distinguish it from the murmur of mitral regurgitation, which becomes fainter with the inspiratory filling of the superjacent lung tissue. In the event of marked regurgitation, a very loud murmur, high venous pressure or right ventricular failure, the inspiratory accentuation of the murmur of tricuspid regurgitation may not occur.

The murmur of tricuspid regurgitation may arise transitorily in case of acute pulmonary infarction.

**3 VENTRICULAR SEPTAL DEFECT** Characteristically, the murmur of ventricular septal defect is a loud, long and harsh pansystolic murmur (sometimes even spilling into diastole), frequently accompanied by a thrill, usually maximal

in the third and fourth left interspaces, heard all over the precordium below the left scapula, and with the second sound either normal or masked. In about half the patients a mid-diastolic murmur appears with increased flow across the mitral valve. Such a murmur requires a flow of more than twice the systemic. It is best heard at the apex.

With a very high septal defect, the murmur may be maximal in the left interspace and accompanied (at times) by an early diastolic murmur at the base (due to incompetency of the medial cusp of the aortic valve). A double murmur simulating the systolico-diastolic murmur of patent ductus arteriosus may be present.

The pansystolic nature of the loud murmur, in an average case with normal pulmonary vascular resistance, is due to the fact that throughout systole the left ventricular pressure is higher than the right. Such a murmur resembles that of mitral regurgitation but can be distinguished from it by the maximal location being along the lower left sternal border. If the pulmonary flow, in such a case, be great, there is also a systolic pulmonary murmur present (although usually masked by the longer pansystolic murmur of the septal defect).

With a moderate degree of pulmonary hypertension or increased pulmonary vascular resistance, the murmur becomes maximal or limited to early systole, thus simulating aortic stenosis. When the pulmonary hypertension is great the systolic murmur of the septal defect may be minimal or absent (because of the small and slow blood flow through the defect) and associated with an ejection click in the pulmonary area, as in aortic stenosis with pulmonary artery dilatation. In rare cases, even without pulmonary hypertension, the murmur may be early systolic in ventricular septal defect, probably due to the defect being confined to the muscular part of the septum and hence closed during ventricular contraction.

**4 ACUTE MITRAL INSUFFICIENCY** This results from rupture of the chordae tendinae, or perforation of mitral valve leaflets, or is common but serious complication of myocardial infarction or bacterial endocarditis. The recognition of this clinical syndrome is important because of the availability of corrective surgical procedures. The murmur is holosystolic with midsystolic crescendo, or may stop short of the aortic closure sound but always starting with the first sound. Other associated findings are normal sinus rhythm, atrial and ventricular diastolic gallop sounds, a split second heart sound, and a rapidly rising but poorly sustained pulse. The systolic murmur of acute mitral insufficiency may have radiation to the axilla and back, if it is due to prolapse of the anterior leaflet of the mitral valve with flow being directed over the posterior leaflet. Occasionally the murmurs are conducted to the base of the heart and give the impression of simulating aortic stenosis. Prolapse of the posterior leaflet of the mitral valve with flow being directed over the anterior leaflet towards the base of the heart is a possible mechanism.

**5 ACUTE TRICUSPID INSUFFICIENCY** This occurs when the tricuspid valve is destroyed by bacterial endocarditis as seen in heroin addicts. The murmur is holosystolic with midsystolic crescendo, or may stop short of the aortic closure sound but always starting with the first sound. Other associated findings are normal sinus rhythm, atrial and ventricular diastolic gallop sounds, a split second heart sound, and a rapidly rising but poorly sustained pulse. The systolic murmur of acute tricuspid insufficiency may have radiation to the axilla and back, if it is due to prolapse of the anterior leaflet of the mitral valve with flow being directed over the posterior leaflet. Occasionally the murmurs are conducted to the base of the heart and give the impression of simulating aortic stenosis. Prolapse of the posterior leaflet of the mitral valve with flow being directed over the anterior leaflet towards the base of the heart is a possible mechanism.

characteristic inspiratory augmentation seen in right sided regurgitant murmurs and is frequently accompanied by a right atrial sound.

**B Early systolic murmurs.** As mentioned earlier, the murmurs of acute mitral insufficiency and tricuspid insufficiency may not be truly pansystolic but may end in late systole well before aortic or pulmonary closure sound occurs. In some small ventricular septal defects, the murmur may suddenly stop in midsystole due to the fact that as ejection continues and ventricular size decreases, the small defect is sealed and the murmur ceases. Such murmurs are often soft and their recognition is important because they are characteristic of the type of ventricular septal defect which might disappear with age. Early systolic murmurs are sometimes heard in patients with tight mitral stenosis and mild or no mitral regurgitation, the mechanism is not clear.

**C Midsystolic murmurs.** Midsystolic murmurs can occur with mitral regurgitation due to papillary muscle dysfunction. In fact any type of systolic murmur can occur with papillary muscle dysfunction. It may be either intermittent or constant during acute myocardial infarction. It is equally frequent with both anterior and posterior wall myocardial ischemia or infarction.

#### **D Late systolic murmurs.**

(1) **CLICK SYSTOLIC SYNDROME OR FLOPPY VALVE SYNDROME.** The late systolic murmur begins about the middle of systole and may continue up to and through the aortic component of the second sound. It is best heard at the apex and, at times, has a tendency to have a late systolic crescendo. It is often introduced by or accompanied by non-ejection clicks. Detailed studies have localised these acoustic events to mitral valvular and subvalvular structures, late mitral regurgitation occurring due to abnormal billowing or prolapse of one or both mitral valve leaflets (usually the posterior leaflet) into the left atrium. It is postulated that chordal or papillary muscle dysfunction causes prolapse of the leaflet with late mitral regurgitation while the sudden tensing or tautening of slackened chordae possibly causes the click (chordal snap). The clicks have been shown to occur at the time of maximal prolapse of the valve by both cineangiographic and echocardiographic studies. If there is slackening of more than one chorda and to different degrees, then there may be more than one click occurring at different times. Physiological and pharmacological manoeuvres that decrease the end-diastolic volume of the left ventricle such as inspiration, standing, amyl nitrite inhalation, and the straining phase of Valsalva, result in an earlier onset of the click and a longer murmur. On the other hand factors that increase the end-diastolic volume like squatting, post-extrasystolic beats and the release phase of the Valsalva manoeuvre move the click away from the first heart sound and the murmur becomes shorter. Infusion of pressor agents intensifies the murmur. The familial incidence of this syndrome has been well documented.



(2) **COARCTATION OF AORTA** In this condition an ejection murmur arises further down the arterial tree and is displaced in relation to the heart sounds. Collateral vessels such as the internal mammary or scapular arteries are more likely to be responsible than the coarctation itself. A similar murmur is found in pulmonary arterial stenosis.

(3) **HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY (HOCM)** In this condition the onset of outflow obstruction as the ventricle contracts produces a murmur at the left sternal edge in the later part of systole, but since mitral regurgitation occurs at the same time there may be other factors involved in the production of the murmur (HOCM is characterised pathologically by asymmetrical or rarely diffuse hypertrophy of the ventricular muscle). It is generally accepted that the apposition of the hypertrophied septum with anterior leaflet of the mitral valve, which may itself be displaced anteriorly due to excessive contraction of the papillary muscle, causes the outflow tract obstruction at the same time permitting mitral regurgitation. A significant amount of stroke volume is ejected more rapidly than normal during the early phase of systole and this is responsible for a flow murmur in early systole (and a rapid upstroke of the carotid pulse). A significant pressure gradient then develops with the flow diminishing as the outflow tract narrows. There is a mid-systolic murmur during this period. When the flow murmur is absent, the midsystolic murmur has a relatively late onset after the first heart sound. At the very end of systole, the gradient may persist with virtually no flow and absence of a murmur.

**E Systolic 'Whoop' or 'Honk'** Harvey described a musical apical systolic murmur which he called a 'whoop' because it seemed to simulate the whoop of whooping cough. Rackley called a similar sound a 'precordial honk' because it suggested the honking noise of a goose. The 'precordial honk' or 'whoop' is a loud, high-pitched, musical, sonorous, vibratory murmur often intermittent, and best heard at the apex usually in late systole. These murmurs may vary strongly with respiration from beat to beat and examination to examination. They are often preceded by clicks which are often associated with ballooning of the mitral valve, mitral insufficiency, or both.

Similar honking noises, with or without clicks, are also produced by transvenous pacemaker catheters situated across the tricuspid valve due to interference with tricuspid closure.

## DIASTOLIC MURMURS

Murmurs occurring during ventricular diastole are of three types depending upon their time relationships to the heart sounds. However, the two main types of diastolic murmurs are those beginning immediately after the second sound and those with an onset delayed to later in diastole.

CLASSIFICATION OF DIASTOLIC MURMURS

A	Obstruction at A-V valves	}	Mid- or late diastolic or both
	Mitral stenosis		
	Tricuspid stenosis		
	Intracardiac tumors such as atrial myxoma		
	Atrial ball valve thrombus		
	Mitral valvitis (Carey Coombs murmur)		
	Coarctation of aorta	}	
B	Regurgitation across semilunar valves		
	Aortic regurgitation	}	Early diastolic
	Pulmonary regurgitation		
C	Increased flow across A-V valves in absence of stenosis		
	(a) Increased flow across mitral valve in left-to-right shunts	}	Mid-diastolic
	Ventricular septal defect		
	Patent ductus arteriosus		
	(b) Increased flow across tricuspid valve in left-to-right shunt		
	Atrial septal defect		
	Mitral regurgitation		
	Tricuspid regurgitation		
	Austin Flint murmur		
	Ebstein's anomaly		
	Hyperthyroidism		
	Severe anaemia		
	Pulmonary hypertension and cor pulmonale		

A Diastolic murmurs associated with obstruction at A-V valves.

1 MITRAL STENOSIS The diastolic murmur of mitral stenosis is dependent on rapid flow through the stenotic orifice of the valve. Thus, the presence of the murmur indicates that there is a gradient across the valve capable of producing a high velocity of flow and turbulence within the recipient chamber—the left ventricle. Two distinct types of diastolic murmurs are recognizable in a case of mitral stenosis of moderate intensity, the mid-diastolic rumbling murmur and the presystolic crescendo murmur (Fig. 9.22). The two murmurs frequently co-exist or are blended together in well-developed cases of mitral stenosis, giving rise to a single, prolonged, mid- and late diastolic murmur, and ending in an accentuated first sound. When the stenosis is more severe

and a large gradient exists across the valve throughout diastole, the mid-diastolic and presystolic (atrial systolic) portions of the murmur merge into one long murmur continuing from the opening snap to the next first heart sound

The atrial systolic phase of the murmur of mitral stenosis provides the characteristic crescendo effect. The typical feature of the murmur is however dependent on the presence of normal sinus rhythm, with a normal P-R interval. In such cases, the atrial systolic portion of the murmur is terminated by the closure of the mitral valve and the first heart sound so that one hears only the crescendo phase. If the P-R interval is prolonged, the atrial systolic murmur runs its full course and will be perceived to be a crescendo-decrescendo murmur.

When atrial fibrillation complicates mitral stenosis, the atrial systolic portion of the murmur drops out and only the middiastolic portion of the murmur persists. Whether the murmur is actually early, middiastolic, late or holo-diastolic depends on the duration of the effective gradient across the valve and the rapidity of the heart rate. The murmur begins immediately after the opening snap of the mitral valve and may continue to the next first sound, giving the impression of a crescendo murmur even in atrial fibrillation. In mild stenosis, however, or during long diastoles, the gradient across the valve is reduced resulting in diminution or disappearance of the murmur in the latter part of diastole.

In mitral stenosis with extreme degrees of pulmonary vascular resistance and pulmonary hypertension alterations in physical signs occur. The peripheral arterial pulse is small due to the low cardiac output. The first heart sound and opening snap may be less loud because of calcification of the mitral valve which interferes with its mobility. The reduction in cardiac output leads to a diminished pressure gradient across the valve despite severe stenosis. Thus, the murmur—both the middiastolic and presystolic phases—may be much reduced in intensity. Tricuspid regurgitation may become a prominent feature.

The mid-diastolic rumble of mitral stenosis has been aptly compared to "the thunder of a distant ox cart rumbling over a loose wooden bridge", "the sound of clearing of one's throat in the bass register" or "the sound of a bowling ball racing down the alley".

Between the second heart sound (indicative of closure of the aortic and pulmonary valves) and the mid-diastolic murmur of mitral stenosis, there is a fairly long and characteristic pause or interval, the duration of which depends on the pressure gradient or relationship between the left atrium and left ventricle, the murmur starts only when the pressure in the ventricle falls below that of the auricle. The diastolic murmur usually starts a fraction of a second after the mitral snap (when present), since the snap corresponds to the onset and the murmur to the completion of opening of the mitral valve.

The existence of severe mitral stenosis without a diastolic murmur is possible. The most common cause of "silent" mitral stenosis is inability to hear the murmur due to a noisy environment or failure to listen with the bell end-piece or with the patient lying on the left side after exercise. The area where the

murmur is heard is very small and can be anticipated by palpation, since the murmur is in the same place ordinarily as the left ventricular apical impulse. The causes of truly silent mitral stenosis include extremely mild valvular changes, very severe stenosis complicated by extreme pulmonary vascular resistance as stated above, and emphysema with barrel chest.

**2 TRICUSPID STENOSIS** Tricuspid stenosis as an isolated sequelae of rheumatic fever is rare. It may be seen in conjunction with involvement of rheumatic mitral or aortic valve affection or as part of complicated abnormalities of congenital origin.

The characteristic murmur, either middiastolic or presystolic in timing or both, is heard at the lower left sternal border and may follow a tricuspid opening snap. In adults with tricuspid stenosis, an important physical sign is an exaggerated A wave in the jugular venous pulse.

**3 CAREY COOMBS MURMUR** In acute rheumatic fever an apical middiastolic murmur, soft and low-pitched may be heard. It is caused by oedema and roughening of the valve leaflets and is a prime sign of rheumatic carditis. It is usually transient.

**4 LEFT ATRIAL MYXOMA** A myxoma is the most common benign tumor of the heart. The presenting symptoms are dyspnoea, syncope, emboli and constitutional symptoms such as fever. A short mid-diastolic murmur which frequently varies and often strikingly with changes in posture is commonly audible. A systolic murmur of mitral regurgitation may be heard, and a characteristic late diastolic sound (tumor plop) may be audible. The first heart sound may be split due to delayed mitral valve closure.

**5 BALL THROMBUS** A ball thrombus in the left atrium may sometimes occur in association with mitral valve disease. Obstruction of the mitral valve may then develop as in myxoma.

**B Diastolic murmurs associated with regurgitation across semilunar valves:** An arterial diastolic murmur is usually caused by backward flow of blood from a main arterial trunk into a ventricle, through a regurgitant or incompetent semilunar valve (aortic or pulmonary), during early diastole.

**1 AORTIC REGURGITATION** Usually associated with structural deformity of the aortic valve, the murmur of valvular aortic regurgitation is an early diastolic murmur (Fig 9 22) starting immediately after the second sound (because of the big pressure gradient between the aorta and left ventricle), very promptly attaining its peak or crescendo (almost at the very start), and then diminishing gradually to give rise to a decrescendo murmur. Although usually early diastolic and short, it may extend throughout diastole, the duration is to some extent dependent on the degree of regurgitation. The murmur of aortic incompetence is usually faint or soft, particularly in rheumatic cases, and may therefore be missed unless looked for with special attention to auscultatory technique. Usually high-pitched and blowing, the murmur

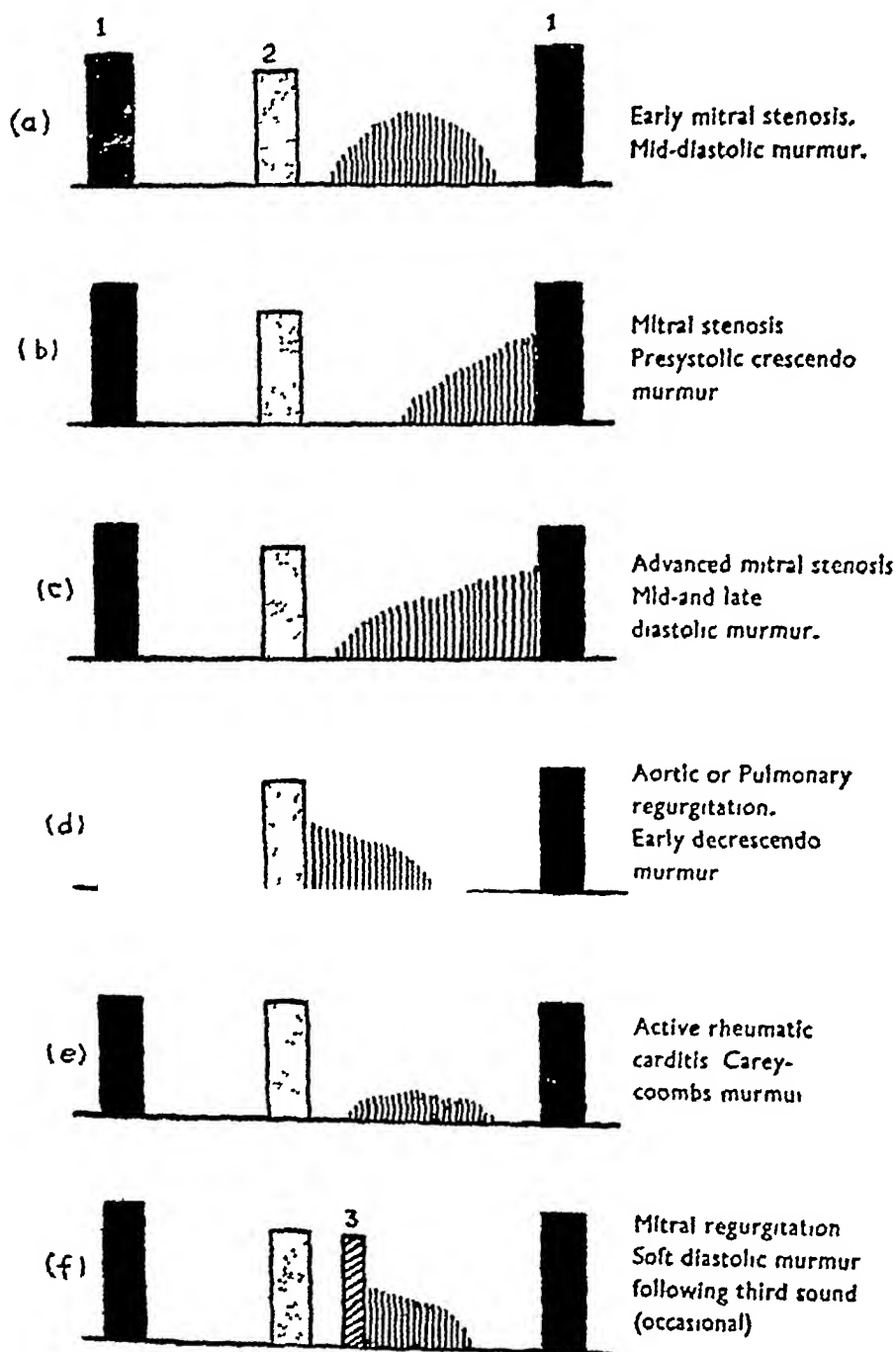


Fig. 9.22 Diastolic cardiac murmurs

has also been described as "sucking", "whiff-like", like the "sound of pouring water" or the "sound of a whispered R" When associated with severe regurgitation, or when transmitted to the apex, the murmur may sound "harsh" or "rasping" When loud, high-pitched and musical (*sea-gull* murmur), it is suggestive of a retroverted or torn cusp The murmur is usually best heard in the third left interspace, close to the sternum, and may even be confined to this area In case of a dilated aorta (as in syphilis), aortic aneurysm, aortic dissection or aneurysm of the sinus of Valsalva, the diastolic murmur may be heard best in the second right interspace Occasionally it is best heard at the apex or the lower end of the sternum Transmission of the murmur is very variable, it may be soft and localized, or loud enough to be heard over the entire precordium or conducted (or localized) to the apical area The murmur may be transmitted downwards, along the right or left border of the sternum, or obliquely downwards towards the apex Being the most high-pitched of all cardiac murmurs, it is best heard with the diaphragm chestpiece (held firmly) or the unaided ear, and with the patient sitting up or bending forward, with arms elevated and breath held in expiration The murmur may be absent (in rare cases) when the valve cusps have become rigid or immobile It is seldom if ever accompanied by a thrill

Associated with the aortic diastolic murmur there is often a moderately loud, ejection systolic murmur, giving rise to the characteristic "seesaw", "to-and-fro" or "bellows" murmur It is due to ejection of an augmented stroke volume across a deformed valve into a dilated initial portion of the aorta Palpation of the carotid pulse enables one to perceive the rapid up-stroke characteristic of aortic regurgitation and thus to exclude the diagnosis of stenosis The murmur is accentuated by squatting, a manoeuvre which may make the murmur audible in a minimal case This posture tends to increase peripheral resistance A similar effect may be obtained with phenylephrine Diminution or disappearance of the murmur may occur in pregnancy due to lowering of peripheral resistance In cases of sudden severe regurgitation, such as might occur in bacterial endocarditis or rupture due to trauma, there is alteration in physical signs The first sound may be absent, probably due to premature closure of the mitral valve resulting from the LV-LA gradient developing in late diastole The diastolic murmur becomes short and harsh, being abbreviated by the damping of regurgitant flow due to the equilibration of pressures in the aorta and left ventricle midway through diastole In aortic regurgitation, the first sound is usually normal, the third sound always absent and the second either accentuated, resonant and tympanitic (as in syphilitic aortitis), or absent (as in aortic stenosis)

The regurgitant flow in aortic regurgitation (compared to that of mitral regurgitation) is very small and of high velocity, resulting in a soft and high-pitched murmur The murmur of aortic regurgitation is best heard (1) with a large Bowles type of chest piece (applied rigidly), (2) during slowing of the

heart rate after exercise, (3) in the correctly positioned patient, and (4) with close auscultatory attention to early diastole

**2. PULMONARY REGURGITATION** The diastolic murmur of pulmonary regurgitation (*Graham-Steell murmur*) is an early diastolic murmur, maximal in the second and third left interspaces, conducted downwards along the left border of the sternum, identical with the murmur of aortic regurgitation in timing, quality and pitch, and usually associated with and somewhat masked by an accentuated second heart sound

The differentiation of this murmur from that of aortic regurgitation may prove extremely difficult, when the peripheral vascular signs of aortic insufficiency are absent or insignificant. In favour of a diastolic murmur being aortic rather than pulmonary in origin, are the high pulse pressure, water-hammer pulse pistol-shot sound, Duroziez's murmur, systolic murmur of aortic stenosis (when present,) wide transmission of the murmur and increased intensity on expiration

While mitral stenosis is the most frequent cause (as recognised by Steell) to cause pulmonary hypertension of such severity as to cause dilatation of the pulmonary artery and incompetence of the valve, a similar mechanism exists in pulmonary hypertension of various other origins, such as *cor pulmonale* and the Eisenmenger syndrome

Pulmonary regurgitation may occur in the absence of pulmonary hypertension from surgical treatment of valvular pulmonic stenosis, SBE, or occasionally as a congenital abnormality. Here the velocity of retrograde blood flow is less, and the murmur is of a lower frequency. This murmur of pulmonary regurgitation in the absence of pulmonary hypertension when sufficiently intense may be transmitted along the left sternal border

### C Diastolic flow murmurs in the absence of stenosis.

*(1) Increased flow across mitral valve in left-to-right shunt* (i) In patent ductus arteriosus, a midsystolic rumbling murmur may be heard at the apex due to a large shunt with an augmented volume of blood returning to the left atrium. The importance of the murmur is to call attention to the large volume of the shunt. (ii) In ventricular septal defect with large left-to-right shunts, the mechanism, as with the large ductus, is a relative stenosis of the mitral valve which is presented with a much augmented flow load. With development of pulmonary hypertension, the apical midsystolic flow murmur may be expected to disappear

*(2) Increased flow across tricuspid valve in left-to-right shunts* (i) In atrial septal defect though the typical murmur is of the systolic ejection type resulting from increased flow through the pulmonary valve, a diastolic murmur is audible in cases with large shunts. Intracardiac phonocardiography has shown that the maximal intensity of the diastolic murmur is in the inflow tract of the right ventricle. This observation supports the theory that the

murmur results from relative stenosis of the normal valve subjected to a very large flow (ii) In mitral or tricuspid regurgitation an early to middiastolic flow rumble is a frequent finding. A middiastolic murmur in M R therefore does not mean the presence of mitral stenosis. The murmur often follows a third sound and is probably caused by vibrations set up by passage of blood into the left ventricle across the mitral valve apparatus stretched taut by the distension of the ventricle following the rapid filling phase. In tricuspid regurgitation, the diastolic flow murmur accompanying the characteristic pansystolic murmur may be a very prominent feature. The diastolic murmur like the systolic murmur may increase in intensity with inspiration.

## CONTINUOUS MURMURS

A murmur which continues throughout the cardiac cycle, extending over both systole and diastole, is referred to as a continuous murmur. It is due to a pressure differential or gradient continuing throughout the cardiac cycle, resulting in a continuous rush or flow of blood.



Fig 9.23 Continuous murmur of patent ductus arteriosus, accentuation in late systole and early diastole

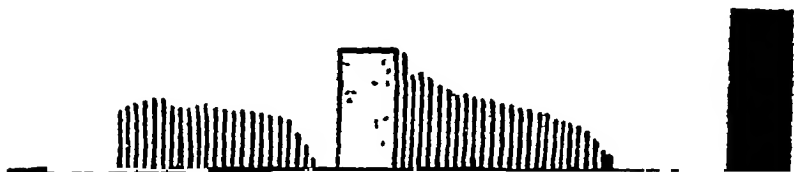


Fig 9.24 To-and-fro murmur of aortic stenosis and incompetence, or of pulmonary stenosis and incompetence

## CLASSIFICATION OF CONTINUOUS MURMURS

A *Shunt between blood vessel or cardiac chamber of high pressure to one of low pressure*

1 Communication between systemic artery and lesser circulation

Intracardiac

Sinus of Valsalva to right atrium, right ventricle or pulmonary artery

Coronary artery aneurysm with fistula to cardiac chamber or pulmonary vessel



Dissecting aortic aneurysm with fistulous communication with right atrium  
 Extracardiac

Patent ductus arteriosus

Post-operative (Blalock-Taussig operation)

Systemic artery-pulmonary vein fistula

2 Communication between pulmonary artery and vein

Pulmonary arterio-venous fistula (congenital or acquired)

B *Blood flow across severely narrowed vessel*

Coarctation of aorta

Aortic arch arteritis (Takayasu's disease)

Peripheral pulmonary artery stenosis (congenital or acquired)

Coronary artery stenosis

C *Increased velocity of blood flow through normal or dilated vessels*

Venous hum

Mammary souffle

Anomalous pulmonary venous return

Bronchial artery dilatation associated with pulmonary atresia, pulmonary hypertension or coarctation of aorta

A Continuous murmurs caused by shunting of blood from high pressure to low pressure By far the most common mechanism associated with continuous murmur is blood flow from a chamber or vessel of high pressure to one of low pressure Such shunting may occur outside the heart between artery and artery, or between artery and vein, or within the heart usually from aorta or coronary artery to a cardiac chamber

#### EXTRACARDIAC SHUNTS

(a) *Persistent ductus arteriosus (PDA)* (Fig 9 23) It was with reference to this entity that Gibson in 1900 first used the term 'Continuous murmur' At birth, because of the high pulmonary artery pressure and low aortic pressure, no murmur or only a systolic murmur is heard The murmur characteristically begins not with, but just after the first heart sound Its intensity is maximum towards the end of systole and early diastole because at that time the difference in pressure in the aorta and pulmonary artery is greatest The murmur is accentuated during expiration and after exercise For some unexplainable reason the typical murmur does not appear usually till the age of three years The murmur then is continuous, best heard in second left intercostal space and under the clavicle, harsh (machinery, humming top, cogwheel, train in tunnel, mill wheel, rolls of thunder or churning) and maximal with the patient recumbent A continuous thrill is commonly palpable When the shunt is large, there is a bounding carotid pulse, and a wide pulse pressure Another useful sign of a large left-to-right shunt is a third heart sound or diastolic flow murmur at the apex due to increased blood flow across the mitral valve in diastole

The diastolic phase of the murmur may disappear in case of pulmonary hypertension recalling the situation in normal infants. In P D A with severe pulmonary hypertension, the systolic murmur may be replaced by an early pulmonic ejection sound. The  $P_2$  becomes accentuated and it may be followed by an early diastolic murmur of pulmonary insufficiency. When a patient has a clinical picture of a left-to-right shunt with pulmonary hypertension and only a systolic murmur at the second or third left intercostal space, ventricular septal defect, atrial septal defect, and aortopulmonary window must also be considered.

The intensity of the ductus murmur is not a dependable guide to the size of the ductus or shunt. The latter can be judged more accurately by attention to the following features: (a) wide pulse pressure and water hammer pulse, (b) a flow murmur at the apex, (c) hypertrophy of the left ventricle.

(b) *Pulmonary arteriovenous and systemic arteriovenous communication* of traumatic or congenital origin, are rare extracardiac causes of continuous murmur heard over the chest.

#### INTRACARDIAC SHUNTS

Intracardiac continuous murmurs occur when there is a fistula between a high pressure vessel or chamber to one of low pressure. Communication may occur between the aorta (sinus of Valsalva) or coronary artery and a cardiac chamber usually the right atrium or right ventricle.

(a) *Sinus of Valsalva aneurysm with rupture*. Most aneurysms of the sinus of Valsalva are thought to develop because of congenital weakness of the supporting tissue. Rupture occurs from the right and noncoronary sinus to the right atrium or ventricle in almost all cases most commonly after the age of 20 years. Therefore, the appearance of a continuous murmur over the precordium in a patient known previously not to have a murmur is suggestive of this mechanism. Chest trauma and severe exertion are the usual precipitating factors in almost all cases. The continuous murmur is heard maximally at the lower left sternal border or xiphoid over the area corresponding to the fistulous tract.

The diastolic accentuation of the murmur is an important sign to differentiate ruptured sinus from patent ductus arteriosus or arteriovenous fistula. Systolic suppression of the murmur is attributed to two factors: mechanical narrowing of the fistulous tract, and a Venturi effect created by rapid ejection of blood past the origin of the fistula.

(b) *Coronary artery aneurysm and fistula*. In almost all cases, there is a communication between a coronary artery and either the right atrium or ventricle. Generally, flow through the coronary arteries is maximal during diastole, therefore the diastolic component of the murmur is louder. Atypical features of the continuous murmur in this condition may be seen when the

shunt goes from a coronary artery to a high pressure R V or to the left ventricle. Then only a diastolic murmur may be heard, since the pressure gradient across the shunt will be obliterated during systole.

The murmur in coronary artery fistula is often superficial and associated with a thrill. Differentiation from extracardiac shunts is usually not difficult due to the location of the murmur on the chest wall, its diastolic accentuation, and alteration with the Valsalva manoeuvre, which decreases the pressure gradient across the intracardiac fistula.

#### B Continuous murmur due to severe arterial stenosis.

Continuous murmurs have been described in association with coarctation of aorta, stenosis of the aortic arch vessels, congenital peripheral pulmonary artery branch stenosis, and acquired pulmonary artery stenosis. The mechanism is related to the high velocity of flow caused by a continuous pressure gradient during systole and diastole across the stenosed vessel. A continuous murmur in pulmonary branch stenosis is due to combination of severe stenosis and large bronchial circulation. The same is true in coarctation where collateral flow through bronchial and internal arteries may be great. It seems that murmurs beginning in late systole and ending in early diastole may occur with stenosis alone in these conditions. However, the longer diastolic components of the continuous murmurs are more likely caused by high velocity flow through collateral vessels.

#### C Continuous murmurs caused by increased flow through normal or dilated vessels

(1) *Venous hum* That increased velocity of blood flow through a normal vessel can produce a continuous murmur is proved by observations on the venous hum. Common in children and less so in young adults, the venous hum is a low or medium pitched, continuous murmur with accentuation in early diastole (because of increased venous flow during the rapid filling phase of ventricular diastole).

Frequently described in the past as a "whine" or "roar" and compared to the "sound of a rope running through a squeaky pulley", the venous hum which is due to the flow of blood through the jugular vein, although widely audible over the anterior part of the neck, is heard best at the lower left border of the right sternocleidomastoid muscle. The murmur is increased in intensity in the erect position, deep inspiration and diastole—factors which result in an increase in flow velocity through the veins of the neck. The murmur is decreased with recumbency, by compression of the neck veins above the stethoscope, by turning the patient's head towards the side of the hum or by Valsalva manoeuvre.

(2) *Mammary souffle* Frequently heard (in about 15 percent) over the intercostal spaces over the sternum on both the sides (more commonly on the left), during the third trimester of pregnancy and the first month of the post-

partum period, the mammary souffle is a soft systolic, to-and-fro or continuous murmur. It results from increased blood flow to the breast. The murmur is loudest when the patient is supine. It may be extremely localised and may be abolished by pressure lateral to the stethoscope.

(3) *Miscellaneous causes* The continuous murmur of anomalous pulmonary venous drainage, bronchial artery dilatation and thyrotoxicosis, as in the case of peripartum breast enlargement, is caused by rapid velocity of flow through dilated vessels. Some tortuosity with slight kinking and therefore minimal or partial obstruction to flow in these vessels is a probable additional factor.

**DIFFERENTIAL DIAGNOSIS** of a continuous murmur depends to a great extent on the familiarity of the clinician with the existence and clinical characteristics of the various causes of continuous murmurs. The first step in diagnosis is confirmation or exclusion of the all-important condition of patent ductus arteriosus. The ultimate diagnosis of the cause of a continuous murmur rests on an appraisal of the entire cardiovascular system in conjunction with a careful study of the various characteristics of the murmur itself and resort to various instrumental diagnostic aids. In the clinical evaluation of a "murmur", from the standpoint of aetiology, the following features are of particular importance.

(1) *Localization of murmur* The site of maximum intensity of the murmur is of great diagnostic value, and must be determined by careful auscultation of the entire chest, back and neck. A murmur maximal in the second or first left interspace is suggestive if not diagnostic of patent ductus, when located above the right or left clavicle, it is suggestive of venous hum or supraclavicular arterial bruit, when low down along the sternum, of coronary arteriovenous fistula or rupture of the sinus of Valsalva into the right side of the heart, a murmur heard better over the back than the front of the chest, suggests truncus arteriosus, pseudotruncus, pulmonary arteriovenous fistula, coarctation of the aorta, congenital pulmonary artery stenosis or constriction of an anomalous pulmonary artery arising from the aorta. The murmur in coronary artery fistula is often superficial and associated with a thrill, the location being at the sternal border. In the case of aortic stenosis with regurgitation and ventricular septal defect with aortic valve defect or insufficiency, the murmur is usually of "seesaw" or "to-and-fro" types and usually heard best in the third or fourth left interspace.

(2) *Past history* of the case is of great importance. Presence of the murmur from birth or early infancy is indicative of a congenital defect. History of a corrective operation (such as Blalock's operation) prior to the discovery of the murmur suggests an artificially induced shunt. History of syphilis in the past may supply a clue to the presence of aortic valve disease.

If the murmur is known to be absent in the past and its appearance associated with severe chest pain or dyspnoea, the possibility of a surgically remediable

condition, such as rupture of the sinus of Valsalva into the right heart, should be considered

A continuous murmur, developing in an individual known to have syphilitic aneurysm of the aorta, suggests rupture of the aneurysm into the pulmonary artery

(3) *Presence or absence or cyanosis* (with or without clubbing) While certain entities, such as patent ductus, aortic septal defect, coronary arterio-venous fistula and coarctation of aorta are acyanotic, others, such as total anomalous pulmonary venous drainage, true truncus arteriosus, pulmonary or tricuspid atresia and severe tetralogy of Fallot are cyanotic

(4) *Resort to instrumental aids* such as fluoroscopy or radiography of the heart in various projections, electrocardiographic study, cardiac catheterization studies and angiography or aortography

**Peripheral Vascular Murmurs** First reported in 1830 by Laennec as "cervical murmurs" of the neck, systolic or continuous (and rarely diastolic) murmurs, of cardiac or extracardiac origin, are fairly commonly heard over the arteries and veins of the neck, when looked for

**VENOUS HUM** This has already been described above

**ARTERIAL MURMURS** These are commonly heard over the large arteries of the neck or extremities (e.g. carotids, brachials and femorals) and may be systolic, diastolic or continuous. They may be completely innocent or indicative of mild or serious organic cardiovascular disease. Routine auscultation over the carotids (cervical vessels) and femoral arteries on the two sides, over the cranium (frontal, parietal and mastoid areas), orbital areas and over the abdomen (renal or splenic areas) may bring to light important auscultatory phenomena

**Carotid murmurs** These may be of several types (1) Physiological or normal murmurs in healthy individuals. These are soft, systolic and localized to the proximal parts of the carotid or subclavian arteries. (2) Systolic or continuous, widely spread or diffuse murmurs are common in hyperdynamic circulatory states, such as severe anaemias, fever, exercise, anoxia, pregnancy, thyrotoxicosis, hepatic failure, cor pulmonale and Paget's disease. (3) Loud systolic murmurs due to aortic or pulmonary stenosis are transmitted well into the neck. (4) Carotid arterial stenosis is frequently associated with a systolic or continuous murmur. With mild obstruction, there is a systolic murmur and with more severe obstruction the murmur becomes continuous. A continuous murmur may also be produced by a carotid artery-jugular vein fistula in which case compression of the vessels should eliminate the murmur and may produce slowing of the heart (Branham's sign). Besides timing of the murmur, ipsilateral and contralateral carotid compression must be practised routine-

ly to evaluate the nature and degree of vascular involvement in such cases. The intracranial and cervical circulation is unique in that anastomotic channels connect the two sides of the circulation through the circle of Willis. Carotid or intracranial stenosis is usually associated with a localized systolic murmur, that is rendered continuous or intensified by compression of the contralateral carotid artery or by light digital pressure over the carotid on the same side. It is usually obliterated by proximal and deep ipsilateral compression of the carotid on the same side.

*Intracranial murmurs* (a) Arteriovenous intracranial fistulae frequently give rise to continuous murmurs with systolic accentuation over the cranium or orbit. Such murmurs can be reduced in intensity and duration or abolished by compression of veins rather than arteries. (b) Angiomas or vascular tumours may give rise to systolic or continuous murmurs.

*Femoral artery murmurs* In aortic incompetence, a systolic murmur may be audible over the femoral artery when compressed. It is due to vibrations set up by the rapid flow of blood past the obstruction. A *diastolic* murmur may be heard over the femoral or brachial artery at times. It is usually intensified by exertion or light pressure over the vessel. In aortic regurgitation (*Duroziez' sign*), a "double-whiff" or diastolic murmur is frequently heard over the femoral artery. It is due to rapid flow of blood toward the heart during diastole and is best heard proximal to the point of compression. A diastolic arterial murmur can also be heard in conditions of peripheral vasodilatation or hyperdynamic circulatory states and as the result of induced vasodilatation after a hot bath in normal healthy individuals.

*Miscellaneous murmurs* Arterial, systolic or continuous murmurs may be heard at times over the gravid uterus, the anterior fontanelle in infants, over the thyroid gland in thyrotoxicosis and over the abdomen in variety of conditions.

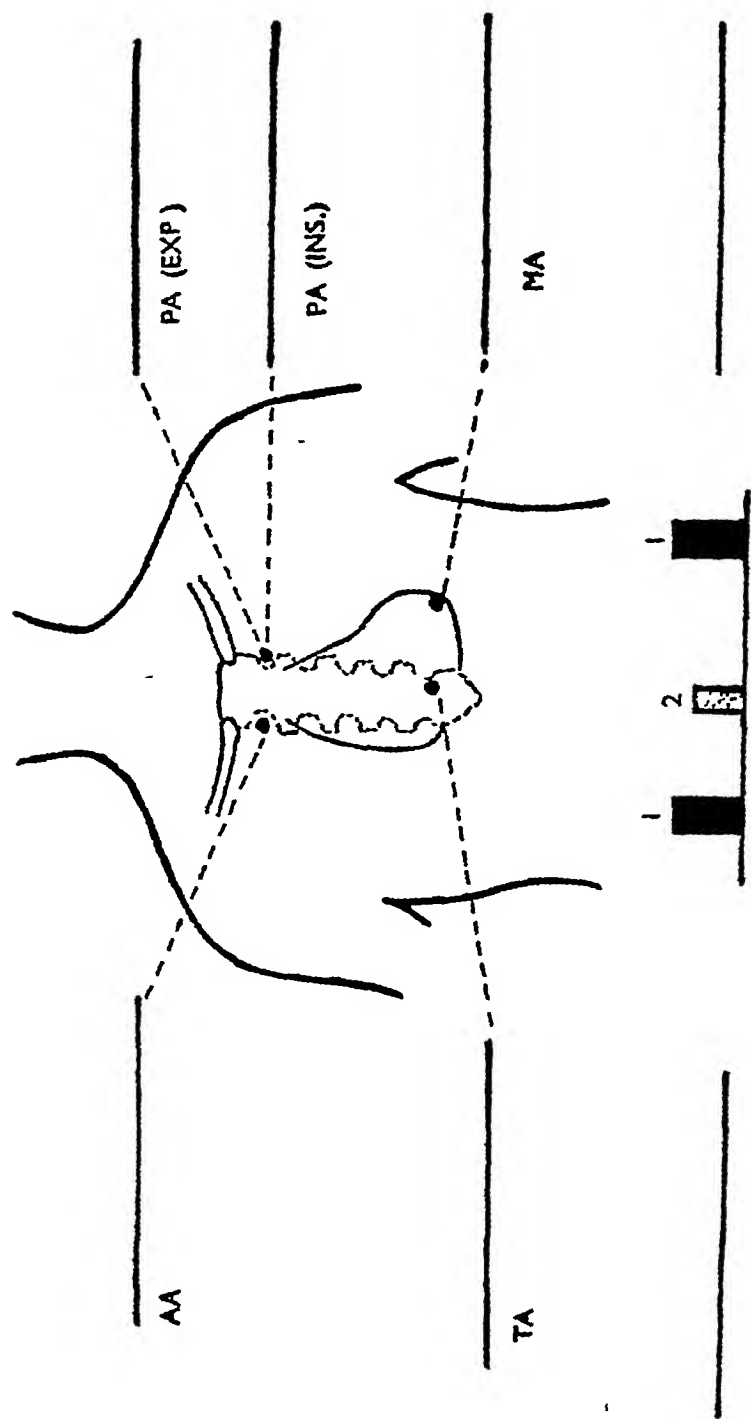
**Graphic recording of auscultatory findings.** Many different methods of recording auscultatory cardiovascular phenomena, such as heart sounds and murmurs, graphically, with the aid of standard charts and symbols have been advocated from time to time, Fig 9 26 illustrates one such method of recording heart sounds and murmurs on a Cardiochart.

Besides involving less time and energy than lengthy written descriptions of auscultatory phenomena, the graphic method of cardiocharting permits a rapid visualization of numerous auscultatory events at a glance and furnishes a permanent control record for future comparisons. The graphic representation of heart sounds and murmurs should be adopted as a routine measure in all cases of valvular heart disease, the process being repeated from time to time to visualize the clinical progress of the case.

RECORD NO.

DATE

NAME OF PATIENT



# STANDARD FOR SOUNDS AT APEX

Fig 9.25 Cardiocharting Chart for recording heart sounds and murmurs Besides the four auscultatory areas—mitral, pulmonary, aortic and tricuspid, auscultatory findings at extra sites such as the third left interspace can be recorded on the lowermost lines The position of the patient during auscultation can be indicated by writing underneath the letters S (sitting), R (recumbent) and LL (left lateral) If the murmur changes on respiration, it should be stated The standard for heart sounds at the apex must be taken into consideration when charting the loudness of the heart sounds in the various auscultatory areas

## PERICARDIAL FRICTION OR FRICTION RUB

This is a variety of exocardial sound which is fairly common. Its importance lies in the fact that (1) it is a valuable diagnostic sign of acute pericarditis, (2) if loud, it may mask or prevent the study of important underlying murmurs, and (3) when confined to one phase of the cardiac cycle, it may be confused with cardiac murmurs.

Pericardial friction rubs are due (1) almost invariably to an acute pericarditis, usually acute fibrinous pericarditis, although at times persisting even with pericardial effusion, (2) occasionally, in thyrotoxicosis, in the pulmonary area, and due to dilated pulmonary artery, (3) occasionally, after pulmonary embolism with acute cor pulmonale, (4) rarely, the rubbing together of normal pericardial layers by pressure or in dehydration, (5) myocardial infarction.

The pericardial or friction rub is caused by the slashing motion imparted to the fibrinous or serofibrinous exudate of pericarditis by the beating of the heart. Some rubs disappear or become less loud owing to pericardial effusion, but many effusions are in fact accompanied by a rub.

**CHARACTERISTICS** of a pericardial rub, sound or friction are: (1) **Quality** This is extremely variable, even in the same case, varying from a soft, blowing or murmur-like sound to an extremely harsh, loud, rubbing, creaking, rasping, leathery, scratchy, shuffling or grating sound. The characteristic rub of pericarditis has been compared to the "creaking of new leather", "grating of nutmeg", "rustling of silk", "rasping of wood" and "crackling of parchment". It may be mimicked by rhythmically stroking the hair above one's ear with one's finger tips. (2) **Timing** The friction sound, although synchronous with the heart beat (thus differing from pleural rub), does not coincide strictly with systole or diastole, it often involves both the phases or "rides astride" both heart sounds. One of the misconceptions about pericardial rubs is that they are necessarily biphasic and it is not uncommon to describe them as "to-and-fro". Biphasic friction rubs usually louder in systole are indeed typical of some cases, and, given the shifting pattern of many rubs may be heard at some time in others. Though there is no single typical pattern of a rub, the most frequently heard is the triphasic rub. The rub occurs with ventricular movement and so is heard in systole, early diastole as the ventricle fills, and again in atrial systole. Much less often the rub may be monophasic. (3) **Site of maximum intensity** The position of the greatest intensity does not correspond to any of the classical valve areas, it is usually best heard near the left edge of the sternum, in the left interspaces. It may be heard better over the base, apex or all over the precordium. (4) The sound is seldom transmitted, remaining localized to a small area, if loud, it may be heard in the back or neck. (5) Its inconstancy is a striking feature. The intensity, character, site of audibility and duration of the rub may vary from day to day. (6) It is usually transitory or evanescent, persisting for a few days or hours only. (7) The intensity varies with the posture, being usually louder



in the upright than in the recumbent position of the patient (8) It is often accentuated by bending or stooping forwards (9) It may be associated with a palpable fremitus or rub on palpation, i.e. felt as well as heard (10) The intensity and clarity of the sound increases with the pressure of the auscultatory chestpiece, particularly in children and thin-chested individuals Because the noise is high-pitched it is necessary to use a diaphragm chest piece pressed firmly to the chest wall against the heart (12) It is usually unaffected by respiration, unlike a pleural rub, which is increased by deep respiration and eliminated by cessation of breathing

#### DIFFERENTIAL DIAGNOSIS OF PERICARDIAL RUB

Pericardial friction rubs have to be differentiated from

1 *Pleural friction* is easy to distinguish as (a) it is independent of cardiac rhythm, (b) conforms to the respiratory movements, and (c) is grossly affected by respiration and cessation of breathing

2 *Pleuro-pericardial friction* may offer some difficulty in diagnosis When pleural friction in dry pleurisy affects the anterior margin of the lung, adjacent to the heart, pleural friction sounds may conform to the cardiac rhythm instead of the respiratory movements Although the sounds occur with the beating of the heart, they disappear (unlike pericardial friction) on deep inspiration, expiration or holding the breath

3 *Heart murmur*, especially when it is confined to one phase of a cardiac cycle (e.g. a systolic monophasic rub), or when "double" (biphasic rub) resembling a to-and-fro double murmur The following are the main distinguishing features (a) Whilst a murmur coincides strictly with systole or diastole of the heart, the pericardial rub does not bear such a clear relationship and may be systolico-diastolic (b) Whilst a murmur is usually blowing, rumbling or musical, a rub is more scratchy, leathery, harsh, or rubbing in character (c) A friction sound is frequently a double sound, unlike a murmur (d) A rub is usually heard best along the left edge of the mid-sternum, and in general not in the areas of maximal intensity typical of most murmurs (e) Whilst transmission or propagation of a murmur is common, such an occurrence is rare in the case of a rub (f) A murmur is usually far more constant in intensity, pitch, character, localization and duration than a rub (g) A murmur is often a persistent or permanent abnormality, a rub is almost always evanescent (h) A rub usually sounds more superficial than a murmur The auditory impression conveyed by a murmur is that it originates in the chestpiece of the stethoscope, whilst a rub appears to originate in the earpieces (i) Pressure on the chest wall with the chestpiece usually accentuates a rub but not a murmur (j) Assumption of the erect position often accentuates a rub, such a relationship may be absent or reversed in the case of a murmur

4 *Other uncommon auscultatory phenomena* (a) "Apex rubs" are systolic, occur in greatly enlarged hearts, and are confined to the precordial area, and,

may, in fact be harsh murmurs (b) "Conus rubs" which are also systolic but confined to the base of the heart occur during acute hyperdynamic states such as pulmonary embolism and thyroid storm. (c) Precordial "crunches" attributed to abnormal chondrocostal and chondrosternal articulations, are sharply localised and permanent (d) Mediastinal emphysema from chest trauma or a ruptured pulmonary bleb can produce a sound similar to a monophasic systolic rub (e) Occasionally, other uncommon lesions produce rub-like sounds (mostly systolic), for example atrial myxoma and A-V valve anomalies such as the floppy valve syndrome

In hydropneumopericardium, a loud churning or splashing sound, the so-called "water wheel murmur", may arise and be audible without a stethoscope

Severe chest pain and pericardial rub frequently coexist in cases of isolated (benign) pericarditis and post-infarction pericarditis. Whilst in the case of the former, the rub appears with the pain at the onset of disease, in myocardial infarction with pericarditis, the rub makes an appearance several days after the onset of pain

#### GUIDE TO SYSTEMATIC AUSCULTATION

Systematic auscultation, according to a pre-arranged or premeditated plan, is essential for correct diagnosis. The examiner, whilst auscultating the heart, should ask himself the following questions

(1) Is the heart slow or fast, regular or irregular? If irregular, is the irregularity phasic (with respiration) or due to dropped beats (extrasystoles or partial heart block) or irregularly irregular (as in atrial fibrillation)?

(2) Is the cardiac rhythm a dual rhythm (with only two sounds per cycle)? If so, is the spacing of sounds normal or tic-tac? What are the characteristics of each sound?

(3) Is there an extra sound present (triple rhythm)? What is the nature of the extra sound? This may be decided on the basis of its precise position in the cycle, its character and the presence of associated signs. Is the extra sound intracardiac or extracardiac?

(4) Are there any murmurs present? What are the characteristics of each murmur? The timing, site of maximum audibility, extent, transmission, intensity, character, pitch, duration, associated thrill, effect of posture, exercise and respiration and relation to the heart sounds must all be investigated separately in the case of each murmur

For a proper study of the heart, auscultation must be practised widely to include not only the classical valve areas, but all other areas (suggested or improvised) that are likely to yield any information. Each patient must be auscultated in the lying down, upright, and, if possible, in the left lateral and bending forward positions, both at rest and after exercise, and also during the different phases of respiration.

A useful plan in auscultation is *selective* auscultation, to mentally split the cardiac cycle into parts. Each heart sound is studied separately and then jointly, the systolic phase and the diastolic phase are then individually studied, to determine the character and time relationships of all extra sounds and murmurs. Finally, all the auditory events are considered collectively, the cardiac cycle being studied as a whole.

**LOCALIZATION OF IMPORTANT CARDIOVASCULAR SOUNDS AND MURMURS** The following *auscultatory areas* or *sites of election*, are capable of reflecting the sites of maximum localization of certain important auscultatory phenomena. Their diagnostic value is therefore unquestioned.

**Apical area (Mitral area)** Sounds usually heard *best* in the "apical area" are the mid- and late-diastolic murmurs of mitral stenosis, the pansystolic murmur of organic or valvular mitral insufficiency (usually transmitted well into the axilla), the systolic murmur of "relative" mitral insufficiency (secondary to the left ventricular enlargement of congestive heart failure, ischaemic or hypertensive heart disease, cardiomyopathy or endocardial fibroelastosis), the presystolic Austin Flint murmur of free aortic insufficiency, the mid-diastolic murmur of left-to-right shunt (as in case of ventricular septal defect, patent ductus arteriosus, aorto-pulmonary fistula or ruptured sinus of Valsalva), and the intermittent diastolic (or presystolic) murmur of left atrial myxoma or ball-valve obstruction of the mitral valve by a thrombus. Also heard *best* in this area are certain insignificant or innocent murmurs, such as the musical, vibratory or twanging-string murmur and the late pleuro-pericardial murmur or click.

Although usually heard *best* elsewhere, certain murmurs are heard *well* enough in the apical area to present diagnostic difficulties. These include the pyramidal or triangular systolic murmur of aortic stenosis, the systolic murmur of congenital cardiac malformations (e.g. septum primum type of atrial septal defect, ventricular septal defect or A-V communis), the loud murmur of septal perforation in case of myocardial infarction and the physiological murmur of hyperdynamic circulatory states (e.g. thyrotoxicosis or severe anaemia).

**Low sternal area (Tricuspid area)** Sounds usually heard *best* in this area are the right ventricular (or protodiastolic) gallop sound of diastolic overloading of the right ventricle (as in tricuspid insufficiency, right-sided heart failure and left-to-right shunt), the right atrial gallop (or fourth) sound, and the opening snap of the tricuspid valve.

Murmurs *best* heard in this area, as a rule, are the diastolic murmur of tricuspid stenosis and the pansystolic murmur of tricuspid insufficiency. Other important murmurs, frequently heard *well* (or even *loudest*) in this area are the pansystolic murmur of ventricular septal defect, the diastolic murmur

of pulmonary insufficiency and the continuous murmurs of coronary arteriovenous fistula, aortic septal defect and ruptured sinus of Valsalva.

*Aortic area* *Sounds and murmurs* usually heard *best* in the aortic area proper (or second right interspace) are the aortic component of the second heart sound, the aortic ejection click or sound of dilatation of the aorta, the pyramidal systolic murmur of aortic stenosis, the systolic murmur of specific aortitis, atheromatous aorta or aortic dilatation, the systolic murmur of free aortic insufficiency, the musical murmur of retroverted aortic cusp and the insignificant or innocent mammary souffle. The early diastolic murmur of aortic insufficiency, the systolic murmur of dissecting aneurysm of the aorta and venous hum, although usually heard best elsewhere, may at times be heard best in the aortic area.

*Pulmonary area* Since a variety of organic and innocent murmurs are encountered in this area, differential diagnosis of auscultatory phenomena often becomes a matter of great difficulty. *Sounds* usually heard *best* in this area are the pulmonary component of the second sound, respiratory splitting of the second heart sound and the pulmonary ejection click or sound of dilated pulmonary artery. The aortic component of the second heart sound and atrial gallop, although usually heard best elsewhere, may be *well* heard in this area.

Murmurs heard best in the pulmonary area include the loud systolic ejection-type murmur of pulmonary stenosis, the soft diastolic murmur of pulmonary insufficiency, the systolic murmur of pulmonary hypertension or dilatation of the pulmonary artery, the continuous machinery murmur of patent ductus arteriosus, the physiological systolic murmur of hyperdynamic circulatory states, the innocent pulmonary systolic ejection murmur and the mammary souffle. The systolic murmurs of certain congenital malformations (e.g. atrial septal defect, aortic septal defect, Fallot's tetralogy and coarctation of aorta), the scratchy murmur of Ebstein's anomaly and the innocuous venous hum, although usually maximal in other areas, may be well heard in the pulmonary area.

*Auxiliary or Additional areas* (1) *Mid-sternal area* (Second Aortic Area) *Murmurs and sounds* usually heard *best* in this area include the early diastolic murmur of aortic insufficiency (probably through the close anatomical proximity of the aortic valve to the chest wall in this area), the systolic murmur of ventricular septal defect (maladie de Roger), the diastolic murmur of Eisenmenger's complex, the systolic murmur of truncus arteriosus, the continuous murmur of coronary arteriovenous fistula, the to-and-fro murmur of aortic stenosis with insufficiency (and of ventricular septal defect with aortic insufficiency) and various pericardial rubs or friction sounds. The sudden appearance of a loud systolic murmur (with thrill) in this area, in case of myocardial infarction, should immediately suggest the complication of a septal perforation or rupture.

Although usually maximal elsewhere, certain sounds and murmurs may be well heard or even best heard in this area. These include the opening snap of the mitral or tricuspid valve, the pulmonary component of the second heart sound, the right atrial gallop sound (as in cases of severe pulmonary hypertension, tight pulmonary stenosis and scleroderma heart), the systolic murmur of atrial septal defect, the continuous murmur of ruptured sinus of Valsalva and the so-called innocent, musical systolic murmur.

(2) *Interscapular area* The interscapular area is often the site of maximum localization of the characteristic systolic murmur of coarctation of the aorta and aneurysm of the descending thoracic aorta. The systolic murmur of dissecting aneurysm of the aorta and of aortic stenosis may also be well heard in this area.

(3) *Infrascapular areas* On either side of the back, below the scapulae and over the bases of the lungs, are the left and right infrascapular areas. Neglected in the past, auscultation of these areas may reveal the presence of important acoustic phenomena, such as the murmurs of the collateral circulation of coarctation of the aorta, the systolic or systolic-diastolic murmur of pulmonary arteriovenous fistula, the systolic murmurs of pseudotruncus arteriosus, Fallot's tetralogy with pulmonary atresia, and pulmonary stenosis and the continuous murmur of truncus arteriosus. The pansystolic murmur of mitral insufficiency may at times be transmitted and heard over the left infrascapular area.

(4) *Cervical areas* The neck region, particularly along the neck vessels or carotid arteries, at the inner ends of the two clavicles, in the supraclavicular fossae and suprasternal notch region, must be auscultated with care in all cases, as this may bring to light important acoustic phenomena.

The systolic murmurs of aortic stenosis, free aortic insufficiency and of dilatation of the aorta (as in atheroma, syphilitic aortitis and aneurysm formation), the systolic murmur of occlusive arterial or cerebro-vascular disease, the systolic murmurs of hyperdynamic circulatory states, the continuous murmur of arteriovenous fistula, the systolic murmur of aneurysms of the innominate, carotid and subclavian artery, and innocent arterial systolic murmurs are often heard well or best over the *carotid vessels*.

Certain murmurs, such as the innocent supraclavicular arterial bruit, the continuous venous hum and the machinery murmur of patent ductus arteriosus, are often heard best over the inner end of one or other *clavicle* or over the *supraclavicular fossa*. Auscultation of the *suprasternal notch* may bring to light the systolic murmur of aortic dilatation, atheroma or aortitis, the accentuated and tambour-like aortic component of the second sound in cases of aortitis or hypertension, or even the opening snap of the mitral valve. Continuous murmurs may be elicited over the thyroid gland in cases of thyrotoxicosis.

(5) *Cranial sites* Auscultation of the skull and orbital regions may bring to light important cardiovascular murmurs. Systolic or continuous murmurs over the skull or orbit are suggestive of occlusive cerebrovascular disease, cerebral arteriovenous fistulae and cerebral angiomas. A pulsating exophthalmos with an audible bruit over the affected eye is almost diagnostic of a carotid artery-cavernous sinus fistula (or rarely an internal carotid artery-ophthalmic vein fistula).

(6) *Abdominal areas* Abdominal auscultation, particularly over the epigastrium, renal areas and flanks, should be undertaken routinely in all cases. This is of particular importance in cases of hypertension of obscure aetiology, where it may bring to light a surgically correctible occlusive disease of one or other renal artery, through the presence of a characteristic murmur. There are numerous causes of systolic, continuous and diastolic murmurs over the abdominal wall. Systemic arteriovenous fistulae (between the aorta and inferior vena cava or between the renal or splenic artery and vein), renal artery stenosis or occlusive disease of the renal artery with hypertension and venous hums (as in cases of cirrhosis of liver, obstruction of portal veins or inferior vena cava and Cruveilhier-Baumgarten syndrome) are the usual causes of continuous or systolic (and rarely diastolic) murmurs over the abdominal areas.

(7) *Miscellaneous areas* Auscultation over the major arteries of the body, such as the femoral, popliteal, brachial and subclavian arteries, may direct attention to murmurs of great diagnostic significance. Thus the presence of "pistol shot sounds" and Duroziez murmur over one or other femoral artery may suggest the existence of aortic insufficiency. A continuous or systolic murmur along one or other major arterial channel may bring to light the presence (and site) of an arterial stenosis, arteriovenous fistula or aortic arch syndrome.

By routine auscultation of such extrathoracic sites, during cardiovascular examination, it is possible nowadays to elicit certain important and surgically correctible diseases of blood vessels, even in the absence of subjective manifestations.

# 10

## The Respiratory System

THE respiratory system, which is concerned with the oxygenation or purification of blood within the lungs, is contained within a conical bony cage, the chest or thorax. Separated from the abdomen below by a musculomembranous partition, the diaphragm, the thorax is formed mainly by the ribs which extend from the spine behind towards the sternum in front on either side. The bony thorax, which is bounded by the clavicles above and the twelfth ribs below, is lined internally by the pleura and externally by skin, fat and muscles. It has an anterior, a posterior and two lateral aspects or surfaces, its anteroposterior diameter being about three-fourths the transverse diameter. All structures lodged within the thoracic cage, such as the lungs and heart, are referred to as intrathoracic.

The main components or elements of the respiratory system from above downwards are the upper respiratory (or air) passages, the trachea, bronchi and bronchioles and the right and left lungs, with their numerous blood vessels and nerves.

### IMPORTANT LANDMARKS AND ANATOMIC AREAS OF THE CHEST

**LUDWIG'S ANGLE.** At the junction of the manubrium with the body of the sternum is a well-defined ridge—angle of Louis. The prominence is of some importance because it indicates (1) the anterior level at which the trachea divides into its two main branches, (2) the upper level anteriorly at which the lungs meet, (3) a convenient landmark from which to count the ribs and interspaces, and (4) the upper level of the atria of the heart.

The spine of the seventh cervical vertebra is a prominence, easily identified at the back, a point from which to count the thoracic spines downwards.

Certain other landmarks, both natural and artificial, are helpful in localizing structures or physical signs within the thorax in relation to the chest wall. For this purpose, the most important natural landmarks are the ribs, the sternum, clavicles, scapulae and the vertebral spine. Besides these, certain arbitrary or imaginary landmarks or vertical lines—midsternal, lateral sternal, midclavicular (or mammary), anterior axillary, mid-axillary, posterior axillary, scapular, and midspinal lines can be used to advantage.

With the help of these landmarks, it is possible to map out the following regions or areas over the thorax (Figs 10 1 and 10 2)

<i>Anterior</i>	<i>Lateral</i>	<i>Posterior</i>
Supraclavicular	Axillary	Suprascapular
Clavicular	Infra-axillary	Scapular
Infraclavicular		Interscapular
Mammary (Pectoral)		Infrascapular

**Surface anatomy of lungs and pleura.** The student must be familiar with the surface markings of thoracic projections of the apices, anterior borders and lower borders of the lungs, the limits of the individual lung-lobes and the lower borders or margins of the pleural sacs on either side (Figs 10 3, 10 4, 10 5, 10 6)

**LUNG FISSURES** The main fissure on each side is indicated by drawing a line from the spine of the second dorsal vertebra, downwards and outwards along the fifth rib as it leaves the vertebral column, to the sixth costochondral junction in front. When the scapula is tilted, by putting the hand on the head, the vertebral border lies along the line of the fissure. The extra transverse fissure, on the right side, is indicated by drawing a horizontal line from the sternum, at the level of the fourth costal cartilage, to meet the line of the main fissure laterally, in the midaxillary line, at the level of the fifth rib or interspace.

**PLEURAL SACS** The pleural membrane has a visceral portion which covers the surface of the lung and is deflected to the surface of the lung at the hilum. The limits of the pleural sacs extend to a lower level in the chest, reaching the 8th, 10th and 12th ribs in the midclavicular, midaxillary and scapular lines respectively. On the left side, the levels are the same as on the right, with the exception that the lung and pleura diverge from the midsternal line at a higher level (lower border of the 4th costal cartilage) so as to leave a portion of the heart uncovered, this position constitutes the area of superficial cardiac dullness.

**LUNGS** The apices of the lungs extend from one to one and a half inches above the clavicles. The lower levels of the lungs in the midclavicular, midaxillary and scapular lines are usually the sixth, eighth and tenth ribs respectively. The hilum of the lung lies opposite the spines of the 4th, 5th and 6th thoracic vertebrae, between the midline and the vertebral borders of the scapulae.

**Segmental anatomy** The bronchopulmonary segments are the topographical units of the lung and are of basic importance to the radiologist and surgeon.

A bronchopulmonary segment is a wedge of lung tissue supplied by a single bronchus and corresponding pulmonary artery and vein. For practical purposes each segment is an independent unit with little significant vascular or bronchial communication with adjacent segments. The segments are



somewhat irregular in shape and quite variable in size They are separated by a thin sheet of fibrous tissue The named pulmonary segments are those which are supplied by the primary divisions of the lobar bronchi

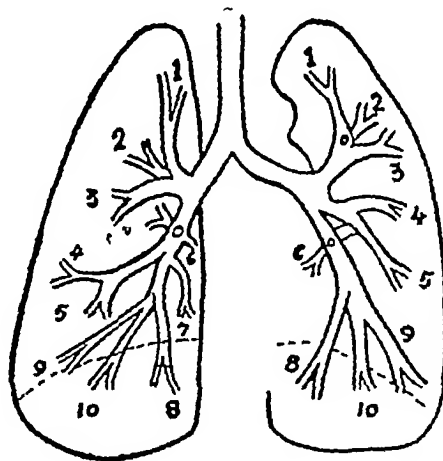


Fig 10 7 Anterior view of the bronchial tree

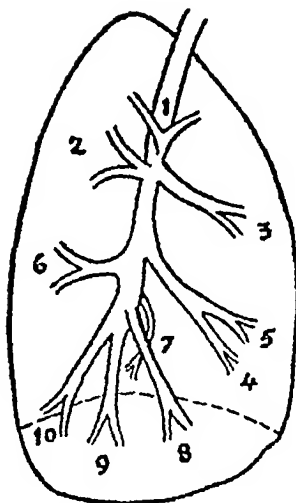


Fig 10 8 Right bronchial tree Right lateral view

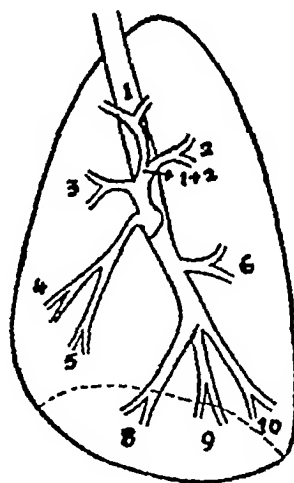


Fig 10.9 Left bronchial tree Left lateral view

The following is the standard nomenclature and numbering of the bronchial tree and broncho-pulmonary segments (Figs 10 7-10 13)

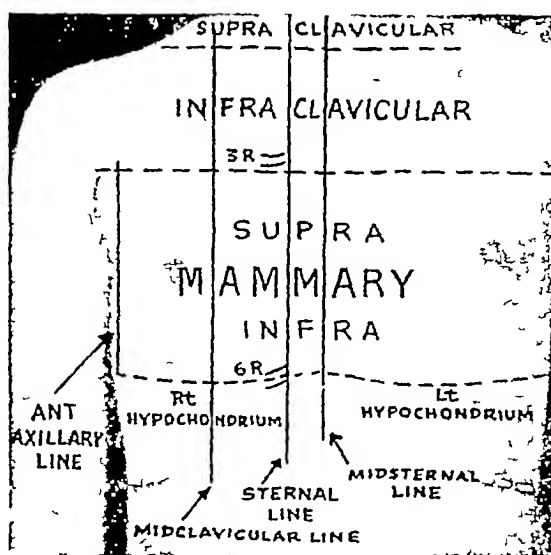


Fig 10.1 Important anatomical landmarks, lines and various areas of the chest. Anterior view

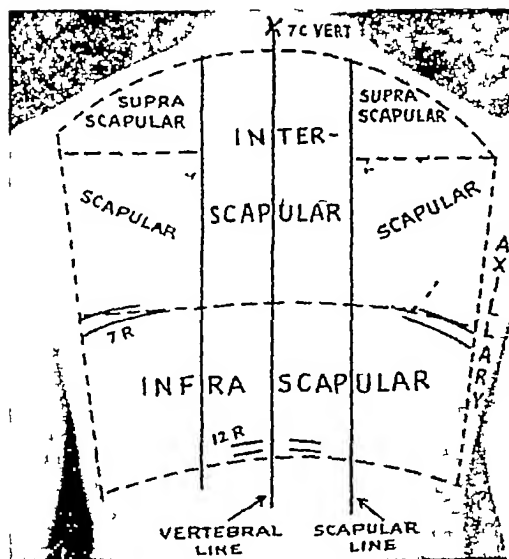


Fig 10.2 Important anatomical landmarks, lines and various areas of the chest. Posterior view

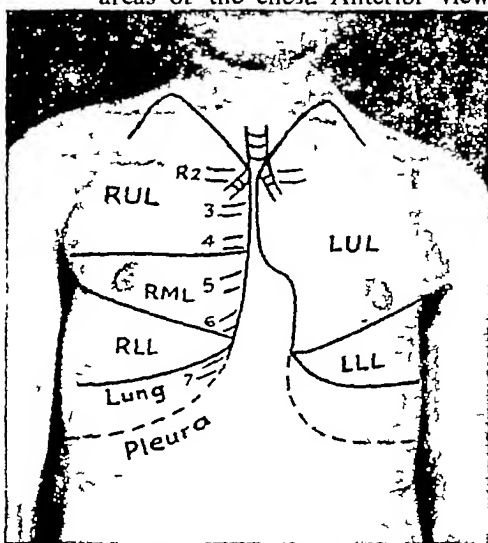


Fig 10.3 Surface markings of lungs and pleura over front of chest

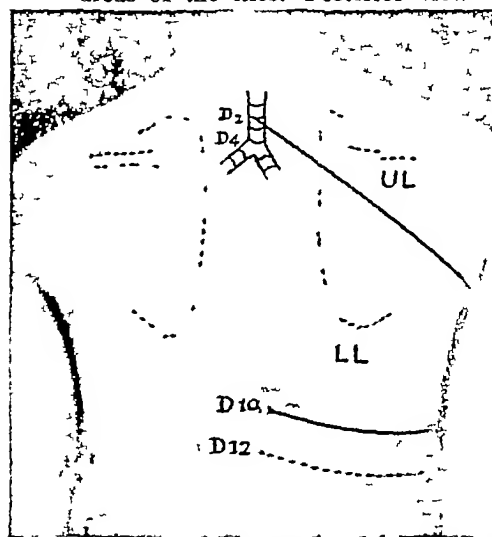


Fig 10.4 Surface markings of lungs and pleura over back of chest

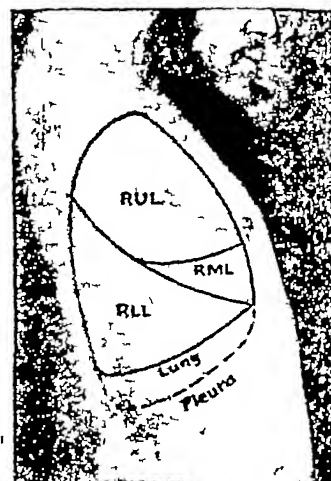
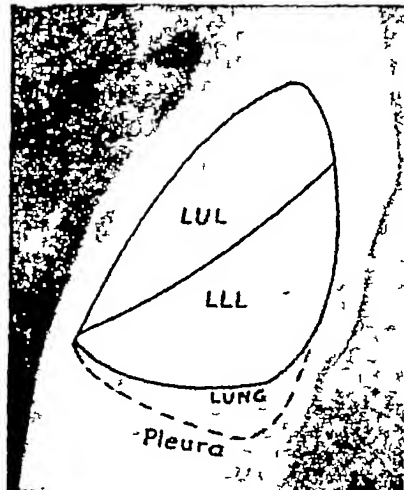


Fig 10.5 Surface markings of right lung and pleura over the right lateral wall of chest

Figs 10.7, 10.8 and 10.9 See p 314

Fig 10.6 Surface markings of left lung and pleura over the left lateral wall of chest



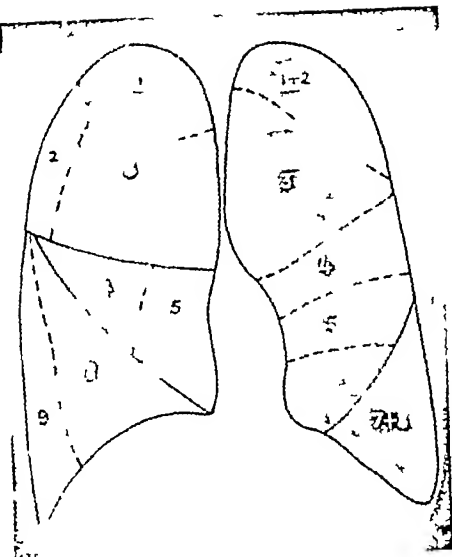


Fig. 10 10 Surface anatomy of pulmonary segments Anterior projection

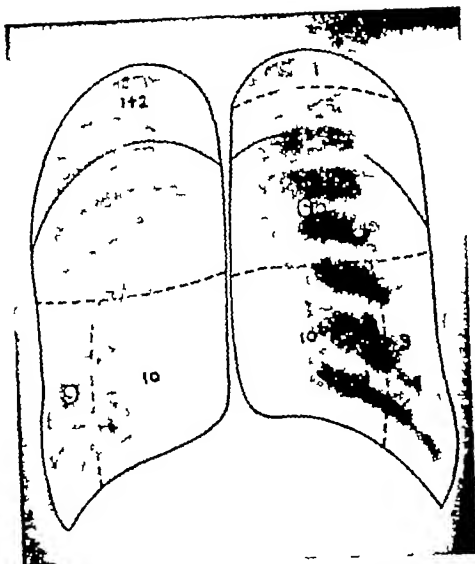


Fig 10 11 Surface anatomy of pulmonary segments Posterior projection

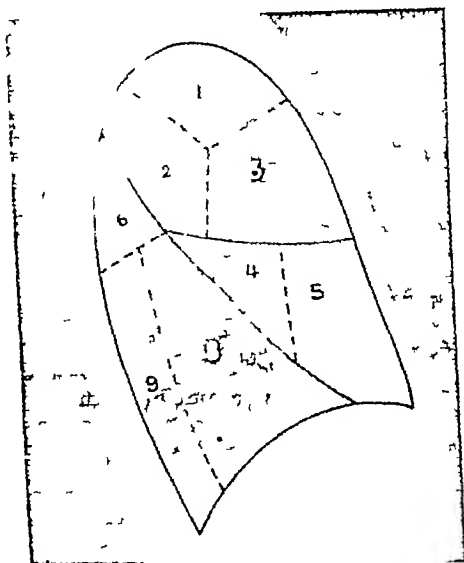


Fig. 10 12 Surface anatomy of pulmonary segments Right lateral projection

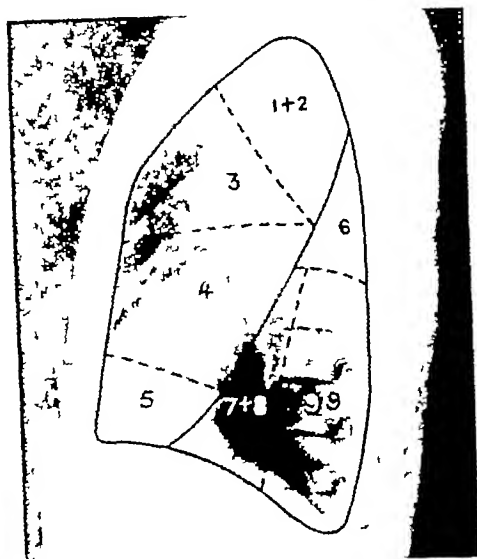


Fig. 10 13 Surface anatomy of pulmonary segments Left lateral projection

*Right lung**Right upper lobe bronchus*

- Apical (1)
- Posterior (2)
- Anterior (3)

*Right middle lobe bronchus*

- Lateral (4)
- Medial (5)

*Right lower lobe bronchus*

- Apical (Superior) (6)
- Medial basal (cardiac) (7)
- Anterior basal (8)
- Lateral basal (9)
- Posterior basal (10)

*Left lung**Left upper lobe bronchus**Upper division*

- Apico-posterior (1 and 2)
- Anterior (3)

*Lingula (lower division)*

- Superior (4)
- Inferior (5)

*Left lower lobe bronchus*

- Apical (Superior) (6)
- Antero-medial basal (7 and 8)

*Lateral basal (9)**Posterior basal (10)*

## AIRWAYS

The trachea commencing at the level of the 6th cervical vertebra descends to divide, a little to the right of the median plane, approximately at the level of the fifth thoracic vertebra to the right main bronchus and the left main bronchus. The point of division of the trachea is known as the carina. Each of the main bronchus gives rise first to lobar and then to segmental bronchi, branching continues within the segment. When considering lung structure it is best to describe the airways by reference to their position in the lung. This is given as the number of generations that separate the given airway from the hilum. The segmental bronchus is counted as the first generation, its first branch and its continuation beyond are the second generation, the next is the third and so on. The proximal five generations are 'large bronchi' because their walls have so much cartilage, between the fifth and fifteenth generations are 'small bronchi' with very sparse cartilage. Bronchioli are those airways distal to the last plate of cartilage and proximal to the alveolar region, there are about ten generations. An acinus is all that lung distal to a terminal bronchiolus. This includes up to eight generations of respiratory bronchioli, alveolar ducts and the alveoli beyond.

**Right Lung** The right main bronchus is divided, for description into two parts—an upper part from the carina to the lower lip of the right upper lobe bronchus and a lower part from this point to the upper lip of the apical bronchus of the right lower lobe, the point where the lower lobe bronchus may be said to commence.

The right upper lobe bronchus arises from the lateral aspect of the upper part of the right main bronchus and after a short course divides into three branches—the Apical, the Posterior and the Anterior—to supply the corresponding segments of the right upper lobe.

The right middle lobe bronchus arises from the antero-lateral aspect of the lower part of the right main bronchus a few centimetres below the origin of the right upper lobe bronchus to divide into a Lateral and a Medial branch supplying the corresponding segments of the right middle lobe.

The right lower lobe bronchus at its commencement gives posteriorly the Apical branch of the lower lobe immediately below the origin of the right middle lobe bronchus. It supplies the apical segment of the lower lobe.

The *medial basal (cardiac) branch* takes its origin from the medial aspect of the lower lobe bronchus a little below the origin of the apical branch to supply a small segment of the lobe near the heart

The next branch lower down is the *Anterior basal* arising from the anterior aspect of the lower lobe bronchus, below the origin of the apical branch. The *Lateral basal* and the *Posterior basal* are the terminal branches of the lower lobe bronchus. These three branches supply the corresponding segments of the right lower lobe.

**Left lung** The left upper lobe is equivalent to the right upper and middle lobes and so the left upper lobe bronchus will have two divisions corresponding to the bronchi of the upper and middle lobes in the right lung.

The upper division after a short upward course divides into two branches, *Apico-Posterior* and *Anterior*. The former also has a short upward course before dividing into the *Apical*, and the *Posterior* branches. *Lingula* or the lower division of the left upper lobe bronchus corresponds to the right middle lobe bronchus. Soon after its origin it divides into the *Superior* and the *Inferior* branches. These five branches supply the corresponding segments of the left upper lobe.

The arrangement of the bronchi and the segments of the left lower lobe is similar to that of the right lung except that there is no branch corresponding to the *medial (cardiac) branch* of the right lower lobe and hence there is no *medial basal (cardiac) segment*.

## INSPECTION

Inspection, next to auscultation, is the most important method of physical examination of the chest. When carried out properly, it yields information of great diagnostic value.

### METHOD

As in the case of the cardiovascular system, for inspection of the chest the patient is stripped to the waist and examined in a good light, daylight being preferred to artificial illumination. In any case, the light must be adequate and fall directly and equally on the two sides of the chest. Inspection of the chest may be carried out in the sitting-up, standing or lying-down position, the first being the position of choice for most cases. It is important that the patient be sitting or lying down absolutely straight. A slant will cause curvature of the spine, which in turn leads to asymmetry of the thorax.

Inspection in the *sitting up* posture is carried out with the patient sitting comfortably on a stool, directly facing the examiner, who is seated on a chair. The *front wall* of the chest is inspected systematically for size, shape, symmetry, respiratory movements, swellings and pulsations, or drooping of one shoulder

After this, the patient is turned over and the *back* similarly inspected, special attention is focussed on the spine for evidence of any curvature, such as scoliosis, which might lead to asymmetry of thorax or abnormal physical signs in the chest and distance of the spine from either scapula. The chest is next examined *in profile*, from one or other side, particularly for evidence of antero-posterior curvature (kyphosis or lordosis) and for anteroposterior diameter or depth. Finally, the upper part of the patient's chest is inspected *from above* downwards (apical or downward inspection). The examiner, standing behind the seated patient, with head bent slightly forwards, looks down over the shoulders at the upper part of the chest. This method of inspection may be valuable in the early detection of apical tuberculosis, yielding as it does an excellent view of the anterior, posterior and upper aspects of the apical regions, any diminution or delay of respiratory excursion, or retraction or flattening of the chest on one or both sides, is suggestive evidence of apical involvement.

In the case of the *recumbent* patient, inspection of the chest is carried out first from the *lower end* of the bed and later from its *side* with the patient lying flat on his back with a pillow under the head. A satisfactory view of the whole of the front wall of the chest can be obtained by observing it tangentially from the lower end of the bed. The upper, middle and lower zones of the chest on the two sides must be inspected individually for size, shape, deformity, bulging, retraction and movements. The chest of the recumbent patient is then inspected *in profile*, from one or other side, the eyes of the seated or kneeling examiner being level with the anterior wall of the chest.

#### SCHEME OF INSPECTION

The following features must be systematically observed, during inspection, in all cases. (1) Size, shape and type of chest. (2) Presence of any asymmetry or deformity of the chest, such as bulging or retraction. (3) Respiratory movements, the rate, rhythm, and type of respiration, abnormalities of respiratory movements on one or both sides, intercostal retraction, movements of the subcostal angle and action of the accessory muscles of respiration must all be looked for. (4) Position of the apical thrust. (5) Diaphragmatic movements. (6) Additional observations, such as enlarged lymphnodes, visible pulsations, sternomastoid sign and oedema of chest wall.

#### SIZE, SHAPE AND TYPE OF CHEST

The normal chest. The normal or healthy adult chest is more or less bi-laterally symmetrical, elliptical in cross-section, the transverse diameter being greater than the anteroposterior diameter in a ratio of 7 to 5 (Fig 10 14), ellipsoidal or conical in shape, the vertical diameter exceeding both anteroposterior and lateral diameters, displays smooth contours, and has a subcostal or epigastric angle of about  $90^{\circ}$ , the angle being more acute in males than in females.

In normal *right handed* individuals, with good musculature, the right side of the chest may appear more prominent than the left, in view of the greater development of the right pectoral group of muscles

In *infancy* and *early childhood* (Fig 10 14), the cross-section of the chest is almost circular, the anteroposterior and lateral diameters being about equal, whilst the general shape of the thorax tends to be cylindrical, the shape of the chest in childhood may be greatly modified by hypertrophied adenoids or rickets

In *old age*, the chest tends to reassume a circular cross-section as in childhood. This is due partly to senile emphysema and partly to degenerative alterations of the bony framework of the thorax

**The abnormal chest** Any abnormality of contour or size of the chest must be noted carefully as it may explain the presence of atypical or abnormal signs on examination, and afford a clue to the nature and extent of present or past disease An abnormal chest may be either symmetrically or asymmetrically abnormal

#### SYMMETRICAL TYPES OF ABNORMAL CHEST

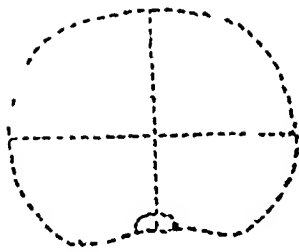
(1) *Long, flat chest* This is an elongated and narrow type of chest, with a reduced anteroposterior diameter, the sternum being closer to the spine than usual. The "flatness" of the chest depends on a loss of the normal forward curvature or angulation of the costal cartilages. A flat chest is usually associated with a long neck, prominent larynx and clavicles, exaggerated supra and infra-clavicular fossae, narrow intercostal spaces and acute subcostal angle. It may be secondary to rickets in childhood, chronic nasal obstruction from hypertrophied adenoids, or bilateral pulmonary tuberculosis

(2) *Alar chest* (Pterygoid chest, "winged" chest) Frequently associated with the long, flat type of chest, the alar chest is characterized by an undue prominence of the vertebral borders of the scapulae, giving a "winged" or "cupid-like" appearance to the chest. It is due to increased obliquity of the ribs

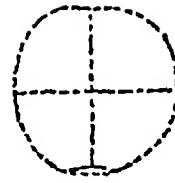
(3) *Phthinoid chest* (Phthisical or pre-tuberculous chest, paralytic thorax) A combination of the alar and flat types of chest, frequently referred to as phthinoid chest, was once regarded as a premonitory sign of pulmonary tuberculosis. It is now known that pulmonary tuberculosis may be associated with any type of thoracic configuration, and that the so-called phthinoid chest is more likely to be the result rather than the cause of tuberculous infection

(4) *Visceroptotic chest* This term has been applied to the long flat chest when associated with visceroptosis or enteroptosis

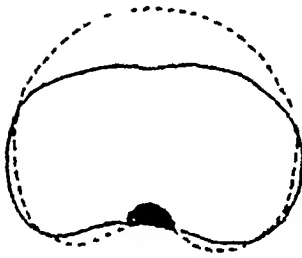
(5) *Pigeon breast* (Chicken breast, keel breast) (Fig 10 15) In the case of the pigeon breast, there is a marked forward protrusion of the sternum and adjacent costal cartilages. In the case of the keel breast, the sternum bulges



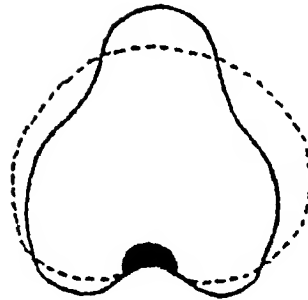
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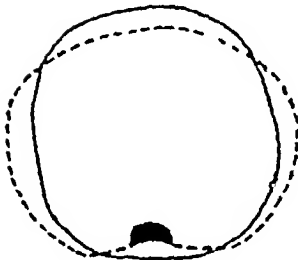
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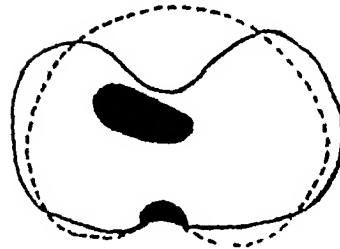
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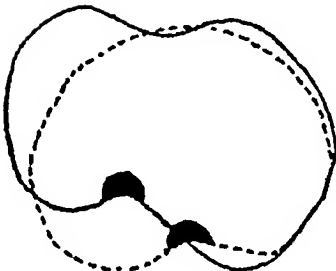
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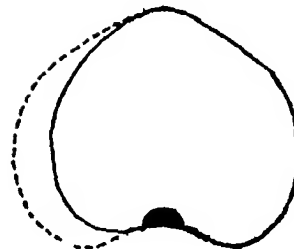
**E**



**F**



**G**



**H**

**Fig 10 14** Cross-sections of normal and abnormal forms of chest  
**A** Adult chest **B** Infant chest (cylindrical) **C** Flat chest **D** Pigeon chest (rachitic) **E** Barrel-shaped or emphysematous **F** Funnel breast, the apex beat moves outwards **G** Kyphoscoliotic chest **H** Unilateral retraction of chest



forward like the "bow" of a ship" and is associated with straightening of the front portions of the ribs and diminished curvature of the ribs at the costal angles in cross-section (Fig. 10-14). Such a chest appears triangular rather than elliptical. Both pectus brevis and pectus excavatum may arise either as congenital anomalies or secondarily to chronic nasal or nasopharyngeal obstruction or rickets in childhood.

(6) Pectus excavatum (Rachitic chest). In childhood rickets, the bones are weak, soft and liable to yield to various pressure and pull. Occultatory factors which predispose to rickets deformities are defective air entry into the air passages and lungs (tends to increase pressure within the chest) and the pull of the various diaphragmatic attachments. More than one variety of deformity is usually associated with the rachitic chest. The most important are: (1) Pectus excavatum with a forward protrusion of the sternum. It has been called pectus carinatum with a forward protrusion of the sternum and straightening or straightening of the anterior portions of the ribs. (2) Harrison's sulci extending away from, as grooves or depressions from the sides of the sternum on each side giving the thorax an appearance of transverse constriction. Harrison's grooves correspond to the costal attachments of the diaphragm and are due to the pulling action of the so-called three attachments. (3) Vertical grooves or longitudinal depressions on each side of the sternum due to pulling of the so-called ribs of the ribs against a pressure within the chest. (4) Rachitic rostrum (Fig. 10-16) bony enlargement of the costochondral articulations particularly of the fourth, fifth and sixth ribs which give rise to two rows of visible and palpable knobs, one on each side of the sternum. The knobs or "beads" in the upper part of the thorax are close to the sternum while the lower ones are situated more laterally. (5) A transverse depression of the lower end of the sternum may be associated with the rachitic type of chest.

(7) Bowry chest (Emphysematous chest) (Fig. 10-17A). In order to accommodate the increased bulk of the lungs in hyperinflated emphysema the thoracic cage becomes cylindrical or barrel-like and displays a circular cross-section (Fig. 10-14). The anteroposterior diameter of the chest is increased, the ribs run more horizontally, the lower ribs tend to flare upwards, the sternum is arched, the angle of Louis is usually prominent, the upper thoracic spine is hyperlordotic, the subcostal angle is wide and the chest remains more or less constant in a state of inflation or inspiratory expansion. The accessory muscles of respiration are usually weak, prominent, and hypercontracted, the shoulders appear elevated, the neck short and thick and the scapular angles become prominent and enlarged. Although usually associated with the hyperinflated emphysema of middle-aged or elderly males, a barrel-chest may be due to old age or senility, severe kyphotic deformity of spine or chronic contact with breastmilk.

(8) Funnel breast (Funnel chest, pectus excavatum, cobbler's breast) (Fig 1018) Exaggeration of the normal hollow over the lower end of the sternum may be a familial or hereditary characteristic, an occupational deformity, as in cobblers or secondary to rickets in childhood. Depending on the degree of deformity, a funnel breast may be described as being of saucer type, cup type or funnel type. This type of chest deformity is clinically of no significance, unless it is severe enough to compress or displace the heart to one or other side. The congenital variety of funnel breast is due to defective ossification of the lower segments of the sternum or to congenital malformation of the diaphragm. The occupational variety, in cobblers, is due to the constant pressure of the shoe against the lower part of the sternum, resulting in a caving-in of this part of the chest.

(9) Gutter breast (Gutter chest) This is a rare variety of congenital deformity of the chest characterized by a shallow vertical groove along the mid-sternal line. It is due to forward convexity of the costal cartilages associated with depression or retraction of the sternum.

(10) Fissured chest This is another rare variety of congenital deformity of the chest characterized by either a deep vertical fissure along the sternum (split sternum or sternal fissure) or a lateral fissure on either side, it is, at times, associated with ectopia cordis or herniation of the lung.

(11) Scaphoid chest (Boat-shaped chest) This is characterized by a depression or hollowing of the anterior aspect of the chest wall, usually from the top of the sternum to the level of the fifth or sixth rib. It may be either post-traumatic or associated with rickets or syringomyelia.

(12) Scorbutic rosary In scurvy, a sharp angulation, with or without beading or rosary formation of the ribs, may arise as the result of backward displacement or "pushing in" of the sternum. The "scorbutic rosary" may be distinguished from the much more common "rachitic rosary" by the sharper angulation of the ribs, and the backward rather than forward displacement of the sternum.

(13) Flail chest (Stove-in chest) A rare form of atypically deformed chest, with paradoxical movement, the flail chest is usually secondary to multiple rib fractures, with or without associated fracture of the sternum.

(14) Shield-like chest A broad chest with widely placed nipples is one of the more consistent features of Turner's syndrome.

(15) Bilaterally contracted chest A bilateral retraction or sinking-in of the chest, resulting in an extreme form of flat chest, may be associated with advanced bilateral pulmonary tuberculosis, lung fibrosis or extreme malnutrition.

(16) Bilaterally expanded chest A symmetrical and extensive bulging or prominence of the two sides of the chest may be associated with emphysema, long-standing bronchial asthma, bilateral pleural effusion or bilateral partial pneumothorax.

ASYMMETRICAL TYPES OF ABNORMAL CHEST, Asymmetry of the thorax may be secondary to (1) deformity of spine, (2) unilateral bulging or prominence of chest, (3) unilateral flattening or retraction of chest, (4) local retraction or shrinking of part of the chest wall.

As a rule, unilateral or localized bulgings of the chest wall are encountered more frequently than unilateral or localized retractions.

It is not always possible to determine which side of the chest is diseased by merely observing the contour of the thorax. This can be ascertained more simply and accurately by comparing the movements of the two sides of the chest during forced breathing. The side which displays less movement or no movement is almost invariably the affected side.

(1) Spinal curvature Asymmetry of the thorax is a common and confusing accompaniment of scoliotic or kyphoscoliotic deformity of the spine (Figs 10 14, 10 19). It is capable of giving rise to abnormal physical signs, through displacement, compression or kinking of intrathoracic organs, such as the heart, lungs and blood vessels.

(2) Unilateral bulging Prominence of one side of the chest with flattening out of interspaces, although usually associated with pleural diseases such as pleural effusion and pneumothorax, may be due to malignant disease affecting the lung or pleura, or compensatory hypertrophy of one lung.

(3) Unilateral flattening or retraction (Fig 10 20). This may result from unilateral tuberculosis, fibrosis of lung, collapse of lung (secondary to bronchial obstruction), long-standing pleurisy, or unilateral wasting of muscles, as in acute anterior poliomyelitis.

(4) Local bulging A localized prominence or bulging of the chest wall may be due to aortic aneurysm, pericardial effusion, gross cardiac enlargement, empyema pointing outwards through the chest wall (empyema necessitas), malignant disease of lung, mediastinum or chest wall, tuberculosis of rib or sternum (Fig. 10 21), spinal deformity, surgical emphysema, or liver abscess (Fig 10 22).

(5) Local retraction A localized flattening or shrinking-in of the chest wall may be due to apical tuberculosis, fibrosis of lung or old-standing pleurisy. Undue retraction or hollowing of the supra-clavicular or infraclavicular fossa (Morenheim's fossa) on one side is suggestive of apical pulmonary tuberculosis.

## RESPIRATORY MOVEMENTS

Normal respiration In health, the rate of respiration in adults is about 18 to 22 per minute, it is somewhat higher in female subjects. It is higher in childhood and infancy, being 40 or more per minute at birth. The rate of respiration is greatly affected, even in health, by exertion, excitement and emotion. The rhythm of respiration is normally regular, but may become irregular during excitement, emotion or self-consciousness, whilst periodic breathing is common during the sleep of old age.

Normal inspiration is an active process, dependent on muscular action of the intercostal muscles, diaphragm and abdominal muscles, and associated with upward and outward movement of the ribs, resulting in expansion and upward movement of the thorax. Normal expiration, slightly longer in duration than inspiration, on the other hand, is a passive process, associated with inward and downward movement of the ribs and a falling-in of the chest walls.



Fig 10 15 Pigeon breast chest



Fig 10 16 Rickety rosary

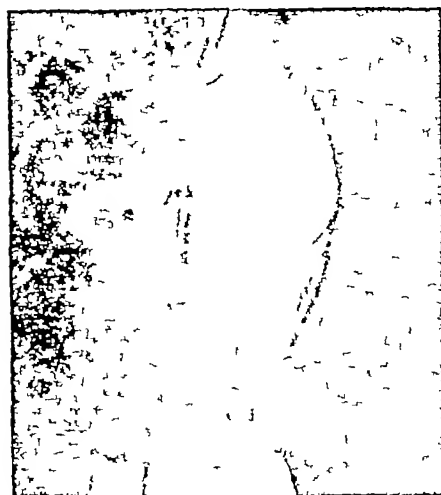


Fig 10 17 Barrel or emphysematous chest.

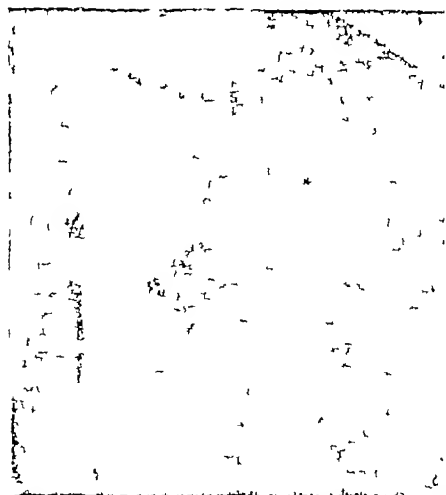


Fig. 10 18 Funnel breast (pectus excavatum)

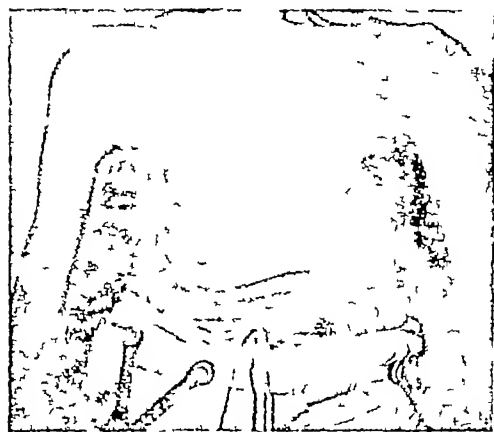


Fig 10 19 Asymmetry of chest due to kyphoscoliosis



Fig. 10 20 Unilateral flattening of c due to fibrosis of lung

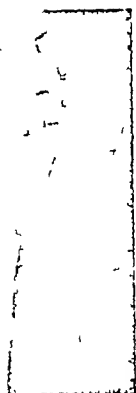


Fig 10.21 Cold abscess of sternum  
causing local bulging of chest wall

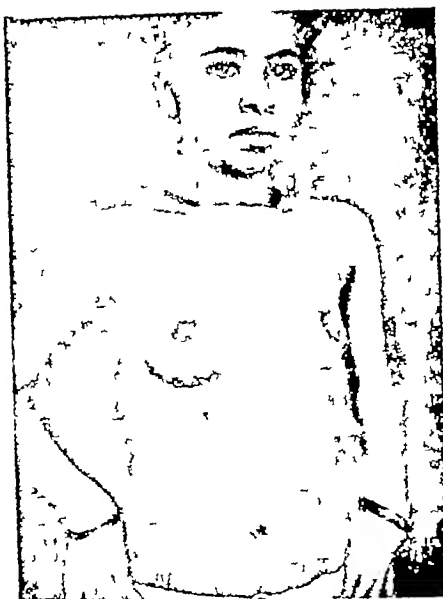


Fig 10.22 Local bulging of chest wall due to liver abscess

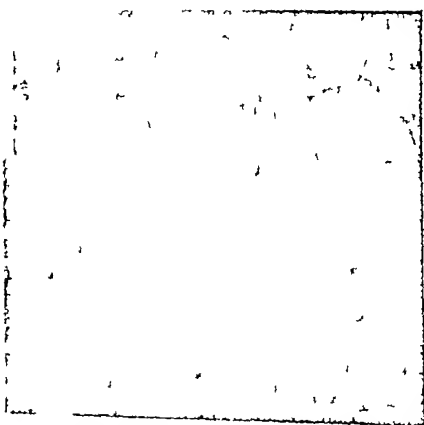


Fig 10.23 Distended veins on chest wall mediastinal tumour

Figs 10.24 to 10.32. See  
pp 336 367

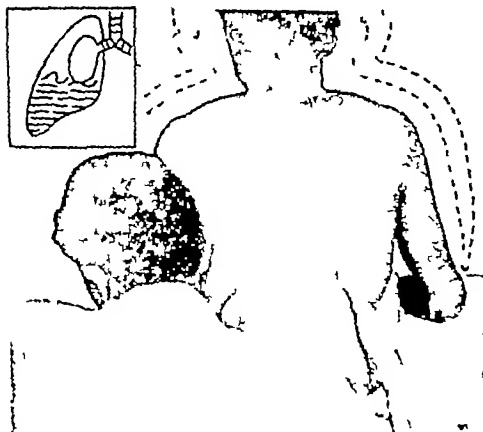


Fig 10.34 Method of eliciting succussion splash

The respiration-pulse ratio, first described in ancient China, is normally about 1 to 4 (18 respirations to about 72 pulse beats per minute)

The type of breathing, in health, depends greatly on the sex and age of the patient. In women, the intercostal muscles play a dominant role in respiration, the respiratory movements mainly involve the upper part of the thorax, the type of breathing being described as thoracic, thoracico-abdominal or costal. In men and children, respiration is mostly abdominal and dependent on the diaphragm, the type of breathing being described as abdominal, abdomino-thoracic or diaphragmatic.

Normally, during respiration, the two sides of the chest move equally, symmetrically and simultaneously, the expansile and upward movement of the chest walls being associated with a mild degree of inspiratory retraction of the lower intercostal spaces and symmetrical outward movement of the subcostal margins on the two sides.

### Abnormalities of respiration

**ABNORMAL RATE AND DEPTH OF RESPIRATION** An increase in respiratory rate may be physiological, as with excitement or exertion, or pathological, as in fevers and diseases of the lungs and heart. It may be compensatory as in the case of the painful shallow breathing of pleurisy or peritonitis. An increased respiratory rate *per se* is referred to as polypnoea or tachypnoea. A decreased respiratory rate is encountered in narcotic poisoning, after administration of morphia or opium, in endocrine diseases associated with hypometabolism, in uraemia, diabetic coma, and raised intracranial tension. In chronic bronchitis, asthma or emphysema, the high expiratory flow resistance can be minimised by keeping the expiratory pressure low. This results in a pattern of slow expiration with short rapid inspirations. In conditions where the lungs are poorly distensible (fibrosing alveolitis, interstitial pulmonary oedema) the most economical pattern is rapid shallow breathing.

**ABNORMAL RHYTHM** The regular rhythm of normal respiration may be disturbed physiologically during excitement or emotion. An irregular respiratory rhythm may be noted in meningitis, coma, peripheral circulatory failure, or as a preterminal event in moribund patients.

(a) Periodic breathing is a special variety of respiratory arrhythmia or disorder, where paroxysms of hyperpnoea or deep breathing alternate with phases of apnoea or cessation of breathing. There are two main varieties of periodic breathing. (1) Cheyne-Stokes breathing, a fairly common variety of abnormal breathing, associated with deep sleep, narcotic poisoning or cardiovascular, respiratory, renal or intracranial disease, and characterized by a gradual waxing and waning of respiratory movements during the phases of expiration. (2) Biot's breathing, a rare type of periodic breathing, usually associated with meningitis and characterized by periods of apnoea between several shallow or few deep inspirations. (b) Suspicious breathing or sighing

*respiration* (compulsive sighing) The characteristic feature of this variety of breathing is an illusion of restricted chest expansion and insufficient intake. Each inspiration seems to fall short of its target until the desire to take a deep breath can no longer be resisted. Immediate relief follows these compulsive maximal inspirations, but after a few breaths the cycle begins again. It usually occurs at rest and the complaint is often associated with anxiety and depression. A compulsive sighing also occurs in apparently healthy subjects and sometimes after injuries to the chest wall or diaphragm.

**PROLONGATION OF RESPIRATORY PHASE** The inspiratory or expiratory phase, or both, may display undue prolongation in disease. Obstructive lesions of the upper air passages, particularly of the glottis and larynx, such as laryngeal diphtheria and glottic spasm, are liable to cause undue prolongation of the inspiratory phase of respiration with or without noisy or stridulous breathing. In obstructive lesions, below the level of the larynx, and particularly in bronchial asthma and emphysema, the expiratory phase is unduly prolonged and wheezy, giving rise to the so-called asthmatic breathing or wheezing respiration. Prolongation of both respiratory phases may be noted in acute alcoholism, narcotic poisoning, diabetic coma, uraemia or cerebral tumour, giving rise to the so-called slow breathing or Kussmaul breathing.

**PURSED LIP BREATHING** Some patients with severe long-standing airways obstruction seem to breathe more comfortably if they purse their lips or grunt during expiration. Presumably they obtain some advantage from these manoeuvres, such as higher expiratory flow rate in return for less effort. In severe widespread airflow obstruction, dynamic compression of the large airways sets a limit to the expiratory flow rate at a relatively low alveolar to mouth pressure gradient. In such circumstances the alveolar to mouth pressure difference can be kept within the required narrow limits by raising the mouth pressure whenever the alveolar pressure tends to overshoot the optimal level. Expiratory grunting as a result of momentarily closing the vocal cords serves the same purpose by raising the intra-tracheal pressure.

**ALTERED RESPIRATION-PULSE RATIO** The normal respiration to pulse ratio of 1 to 4 may be altered in disease. In case of pneumonia and at times in congestive cardiac failure, especially when associated with heart block, the ratio may become 1 to 3 or 1 to 2. Rarely, in bronchopneumonia the ratio may be 1 to 1. The exact opposite may be noted in case of opium or morphia poisoning, where the ratio may become 1 to 5 or 1 to 6.

**ABNORMAL ABDOMINO-THORACIC BREATHING** An abdomino-thoracic or male type of breathing in a woman, or of thoracico-abdominal or female type of breathing in a man or child should be viewed with concern and investigated fully for the cause of the disorder. Whilst abdominal breathing in a woman is suggestive of thoracic disease, such as acute pleurisy, malignancy, or arthritis of the dorsal spine, the discovery of costal breathing in a man or

child suggests the possibility of intra-abdominal disease, such as ascites, hepatomegaly, splenomegaly, massive tumour, peritonitis or diaphragmatic pleurisy. Exaggeration of normal breathing may also be indicative of disease. For instance, an entirely thoracic type of respiration, instead of the normal thoracico-abdominal type, in a woman is suggestive of intra-abdominal disease or diaphragmatic paralysis, similarly, an entirely abdominal or diaphragmatic type of breathing in a man or child suggests the possibility of dry pleurisy or intercostal muscle paralysis.

**ATAXIC BREATHING** This is an irregular pattern of breathing in which both deep and shallow breaths occur randomly suggestive of a lesion of reticular formation of distal part of medulla.

**APNEUSTIC BREATHING** A rare type of respiration characterised by a pause at full inspiration or more commonly brief end-respiratory pauses lasting 2 or 3 seconds, often alternating with expiratory pauses as well as with other irregularities of respiratory rhythm. Apneustic breathing is suggestive of damage to respiratory control mechanism at pontine level.

**STRIDOR** This is a crowing sound due to obstruction in the larynx or trachea. It is as a rule inspiratory because of more rapid airflow during inspiration. *Laryngeal stridor* is somewhat highpitched and is caused by a foreign body, obstructing the laryngeal orifice, laryngeal spasm, oedema or bilateral paralysis of vocal cords. *Tracheal stridor* is lower in pitch and dyspnoea is always associated with it. It tends to be biphasic. It is increased by coughing. It usually results from obstruction of the lumen of the trachea by a malignant growth. *Bronchial stridor* is a term sometimes used to refer to the wheeze in obstructive airways disease.

**CATCH IN BREATHING** In pleurisy or pneumonia, the respiration may be rapid and shallow and there may be a sudden catch in breathing owing to pleuritic pain if patient takes a deep breath.

**Thoracic movements. METHOD** The movements of the thoracic wall during respiration (normally of the nature of expansion and upward movement) must be carefully observed by inspection, both during quiet and forced or deep breathing, for the following features: (1) depth of breathing or amplitude of movement, on either side, (2) nature of movement, whether expansive or up-and-down movement as in emphysema, (3) asymmetry or inequality of movement, indicative of increase or decrease of movement on one side, (4) asynchronicity of movement due to delayed onset or "lag" of movement on one side, (5) local deficiency of upward movement or expansion, (6) intercostal retraction (during inspiration) or bulging (during expiration), (7) movements of the sub-costal angle and costal margins, and (8) behaviour of the accessory muscles of respiration.

The respiratory movements of symmetrical sections of the chest on the two sides must be compared in the following order: upper or apical zones, middle zones, lower zones, intercostal spaces, and costal margins.



## ABNORMALITIES OF THORACIC MOVEMENTS

(1) *Bilateral diminution of expansion* A symmetrical restriction of movement on the two sides may be due to pulmonary emphysema (where expansive movement of the chest is replaced by up-and-down movement), massive pericardial effusion, large mediastinal tumour, bilateral fibrosis of lungs, arthritis of spine, fractured ribs, or dry pleurisy. Measurement of chest circumference at the nipple level can be carried out before and after a vital capacity manoeuvre. In hyperinflation of the chest due to chronic obstructive bronchitis, the expansion will be restricted to 3 cm or less instead of the normal 5-7 cm.

(2) *Inequality or asynchronicity of movement* When the two sides of the chest move unequally or asynchronously, during quiet or forced breathing, the side that moves less or lags in movement is nearly always the diseased side, a rule that is seldom broken. In case of unilateral intrathoracic disease pulmonary or pleural, the affected side displays both diminished amplitude of movement and slight delay or "lag" of movement.

(3) *Unilateral diminution of expansion* This may be due to lung disease, such as pulmonary tuberculosis, pneumonia and lung collapse or involvement of pleura, as in pleural effusion and pneumothorax. Diminution of movement, limited mainly or solely to the upper or apical zone, suggests pulmonary tuberculosis, preponderant affection of the lower zone suggests pleural effusion, bronchiectasis, fibrosis or collapse of lung.

(4) *Unilateral increase of expansion* Increased respiratory movement on one side is much less common than decrease of movement, it may be due to compensatory emphysema, an attempt on the part of the lung to compensate for decreased activity or collapse of the opposite lung.

(5) *Inspiratory retraction of interspaces* A mild degree of indrawing of the lower interspaces is normal during inspiration, excessive intercostal retraction during inspiration, whether bilateral or unilateral, is indicative of disease. When bilateral it suggests tracheal or laryngeal obstruction as in laryngeal diphtheria or severe bronchial spasm as in bronchial asthma, when unilateral, it suggests pulmonary collapse (secondary to bronchial obstruction) or fibrosis of lung on that side.

(6) *Diminution or absence of normal intercostal retraction, during inspiration* suggests a massive pleural effusion or tension pneumothorax.

(7) *Overaction of accessory muscles* Whenever distended or inelastic lungs require forcible compression for deflation during expiration, as in bronchial asthma and emphysema contraction of the accessory muscles of respiration, namely the sternomastoid, trapezius and other neck muscles, may be observed together with inspiratory indrawing of the suprasternal and supraclavicular fossae due to high negative intrathoracic pressure, and in order to suck more air through the narrowed airways. In course of time the muscles display chronic hypertrophy.

(8) *Movements of costal margins* The lower limit or border of the thoracic cage, anteriorly, is formed by the right and left costal margins meeting at the subcostal, costal or epigastric angle, with the xiphisternal or ensiform cartilage in the centre. Normally, the two costal margins move outward during inspiration. This is because the inward pull of the highly arched normal diaphragm is overpowered by the outward pull of the intercostal muscles.

Inward or paradoxical movement of the costal margin during inspiration may be seen in the presence of substantial airways obstruction and may constitute a useful sign in a breathless patient. Deficient movement of one or both costal margins during inspiration is suggestive of unilateral or bilateral depression of the diaphragm, as a result of massive pericardial effusion, gross cardiac enlargement, pleural effusion, pneumothorax or emphysema. When the diaphragm is flattened or pushed down by disease, its inward pull on the costal margin becomes strong enough to overpower the outward pull of the intercostal muscles.

(9) *Diaphragmatic movements* (Litten's phenomenon, phrenic wave) Once rated high in importance, Litten's sign has been more or less supplanted by fluoroscopy for the study of diaphragmatic movements. The term phrenic wave refers to the up-and-down movement of a shadow, over the axilla from about the level of the seventh to the tenth rib, during respiration, with the recumbent patient directly facing a window and the examiner sitting on one or other usual side with his back to the window.

Restricted excursion or absence of the shadow is suggestive of impaired or abnormal diaphragmatic movement, as in pleural effusion, pneumothorax, lobar pneumonia, pulmonary tuberculosis and subphrenic abscess. In case of hepatomegaly or liver abscess, the behaviour of the shadow is usually normal. Litten's sign is of little or no value, particularly in shallow breathers or obese subjects, in whom the sign may be absent even in health.

#### ADDITIONAL OBSERVATIONS

**Position of apex thrust.** It is important to locate the exact position of the cardiac apex in all cases of respiratory disease, as it may be displaced to one or other side (mediastinal shift) by disease of lung or pleura. As a rule, it is displaced or pulled towards the side of the lesion in case of lung fibrosis or collapse, and away from the side of the lesion or pushed in case of pleural effusion, empyema or pneumothorax, the apex beat remains in its normal position in case of pneumonia.

**Sternomastoid sign** (Trail's sign) Undue prominence of the clavicular head of the sternomastoid muscle, on one side, is usually indicative of tracheal displacement (or mediastinal shift) to that side, as in the case of apical tuberculosis or lung fibrosis.

**MECHANISM** The pretracheal fascia encloses the sternomastoid muscle and is in close contact with the tendon of the muscle. Tracheal displacement is reflected therefore to an increased tension of the sternomastoid muscle (in its tendinous part) on the same side as that to which the mediastinum is pulled or pushed.

**Distended chest veins** Prominent or engorged veins over the front of the chest are suggestive of superior vena caval obstruction, they furnish an early

important sign of mediastinal new growth (Fig 10 23), enlarged mediastinal lymphnodes or aortic aneurysm Small venules along the costal margins (Emphysematous girdle), although regarded once as indicative of emphysema are of no clinical importance

Signs of hyperinflation of the chest Besides reduced expansion of lower ribs and use of accessory muscles of respiration, one may observe that the shoulders are high, the tracheal descent with inspiration is less than normal (probably because of low diaphragm), and there is filling of external jugular veins during expiration

Pulsating vessels. Visible and pulsating subcutaneous vessels along the interspaces and over the back are characteristic of the anastomotic circulation of coarctation of the aorta

Pulsatile bulge A pulsatile swelling anywhere over the chest wall (especially anteriorly) may be caused by an aortic aneurysm, vascular new growth (such as lymphosarcoma) or empyema necessitas

Oedema of chest wall This may be either unilateral, as in empyema, or bilateral as in congestive heart failure or wet beri-beri

Enlarged glands Visible enlargement of lymphnodes in the neck, axilla or supraclavicular fossa is a particularly significant finding and must be investigated carefully

## PALPATION

Palpation, besides confirming the findings of inspection, frequently provides additional information of diagnostic value

**SCHEME OF PALPATION** The following observations must be routinely made during palpation (1) Comparative palpation of respiratory movements on the two sides (2) Diaphragmatic movement. (3) Position of mediastinum (4) Contour of chest. (5) Localized swelling (6) Localized tenderness (7) Tactile vocal fremitus (8) Other varieties of fremitus (9) Thoracic rigidity

Respiratory movements *Comparative palpation* of the two sides of the chest in an orderly manner from above downwards, comparing the extent or amplitude of respiratory movement of the upper, middle and lower zones, may bring to light minor degrees of inequality of movement, otherwise missed on routine inspection

**METHOD** For palpation of the upper zones or apical regions of the lungs the patient sits on a stool with the head bent forwards. The examiner places his two hands over the apical regions from behind, the thumbs being approximated in the midline at the back. The excursion of the thumbs from the midline, as the patient breathes, indicates the degree of expansion on the two

sides If the patient is recumbent, the hands of the examiner are placed over the two clavicles in front The expansion of the apical regions can be assessed by noting the extent to which the hands are lifted during deep inspiration

For palpation of the *middle* and *lower* zones of the lungs, the two hands of the examiner are placed symmetrically, on either side of the patient's chest, so as to grip the two sides with the palms, whilst the thumbs are actively stretched in order to just meet in the midline during expiration The excursion of each thumb from the midline, during deep inspiration, is a fair guide to the degree of expansion of the lung on that side

Comparative palpation of the upper, middle and lower zones is then carried out in a similar manner over the back

Asynchronicity of movement of the thumbs or hands on the two sides, during comparative palpation, is evidence of lag or delay of movement on one side

**Diaphragmatic movement** **METHOD** This is assessed by placing the flat of one hand over the anterior wall of the chest and the other over the epigastrium of the patient Normally, during inspiration, both the examiner's hands are lifted, one by the expanding chest and the other by the rising epigastrium

**SIGNIFICANCE** Immobility of the epigastrium during inspiration is suggestive of intra-abdominal disease, with fixation of the diaphragm A paradoxical retraction or withdrawal of the epigastrium during inspiration is suggestive of diaphragmatic paralysis

**Mediastinal position.** Displacement of the mediastinum to one or other side by disease of lung or pleura may be suggested by alteration in the position of the apical thrust or displacement of the trachea in the suprasternal notch to one or other side

**APEX THRUST** Displacement of the apex thrust can be demonstrated by palpating first with the palm of the hand and later with the fingers to determine its exact site Apex beat deviation (and deviation of cardiac dullness) are the main indications of the shift of the lower mediastinum

**POSITION OF TRACHEA** Displacement of the trachea to one or other side, from its normal midline position in the neck, is an important sign of mediastinal shift or displacement The index finger of the examiner's right hand, when pushed directly backwards in the suprasternal notch, normally encounters the trachea in the midline When the trachea is deviated to one side, the finger slides along the other side of the trachea, the resistance to pressure being less on that side

An alternative method of detecting tracheal deviation is to push the index and middle fingers of the right hand directly backward, on either side of the trachea, in the suprasternal notch The resistance encountered is much greater on the side to which the trachea is displaced

Lateral displacement or deviation of the trachea is the main indicator of lateral shift of the upper mediastinum This may be of diagnostic significance,

especially in post-operative and seriously ill patients. Lateral displacement of the trachea may bring about deceptive or misleading physical signs over the apical region of the lung.

Causes of deviation of mediastinum The mediastinum may be *pulled* due to collapse of lung, fibrosis of lung, or old pleural effusion or empyema (due to failure of re-expansion of lung). It may be *pushed* by pleural effusion, pneumothorax, or a large mass in the lung usually in upper mediastinum.

Forward (or anterior) displacement The trachea may be rarely displaced anteriorly, by tumours, abscesses or diseases of the cervical spine.

TRACHEAL DESCENT WITH INSPIRATION On full inspiration in young normal subjects, the distance between the suprasternal notch and the lower margin of the cricoid cartilage is 3 to 4 finger breadths, the distance diminishes with age. The decrease is particularly marked in patients with airways obstruction owing to elevation of the sternum relative to the hilum, and thus in hyperinflation of the lungs, the length of the trachea palpable above the sternal angle in expiration becomes less. In such patients a finger tip on the thyroid cartilage may demonstrate tracheal descent on inspiration, probably owing to contraction of the depressed diaphragm.

Contour of chest The shape or contour of the chest, although usually obvious on inspection, can be confirmed by palpation either with the examiner's hands or with a special appliance called the cyrtometer or cyrtograph. The instrumental method of delineating or measuring the curves of the chest, *cyrtometry*, is useful for determining the shape of the cross-section of the chest (whether elliptical, triangular or circular) and for detecting asymmetry or deformity of the chest.

Localized swelling Any bulge, swelling\* or prominence of the chest, whether pulsatile or not, must be investigated by palpation for size, shape, localization, tenderness, heat, redness, fluctuation, pulsation and thrill.

Localized tenderness Areas of local tenderness, usually obvious on routine palpation of the chest, may afford diagnostic information. Thus local tenderness over one or more ribs may suggest fracture from coughing or trauma or secondary deposit, or necrosis, over the interspaces, acute pleurisy or empyema, and over the precordium or apical region, neuro-circulatory asthenia.

Tactile vocal fremitus The detection, by palpation or tactile perception, of vibrations, communicated to the chest wall from the larynx via the bronchi and lungs during the act of phonation, is referred to as tactile vocal fremitus (TVF) or vocal fremitus.

METHOD For detection of vocal fremitus, the examiner places either the palm or the ulnar border of his hand depending on individual preference, on some area of the chest wall, whilst the patient is made to repeat some suitable phrase, such as "ninety-nine" or "one two three", over and over again, in a clear voice, keeping the intensity and pitch of tone strictly constant. In view of the unequal sensitivity of the two hands to vibration, the *same hand* must be employed throughout the elicitation of this sign. It is also necessary to compare *symmetrical areas* on the two sides of the chest, proceeding sys-

tematically downwards from the apices to the bases, first anteriorly, then along the axillae, and finally posteriorly. Selection of these particular phrases, for eliciting vocal fremitus, depends on their ability to bring out the resonance of the voice in a most satisfactory manner.

For the production and perception of vocal fremitus, two conditions are essential (1) the patient's voice must be sufficiently resonant, and (2) the bronchi, supplying the part of the lung under investigation must be patent.

PHYSIOLOGICAL VARIATIONS Even in perfectly normal, healthy subjects, marked variations of vocal fremitus are common. The degree of fremitus depends on a variety of factors, such as age, sex, race, volume and pitch of voice, thickness of chest wall, constitution, state of nutrition and site of chest wall palpated. Vocal fremitus is usually better felt in males than females, because of the greater volume and deeper pitch of the voice in the former. As a rule, fremitus is less marked in children than adults. Negroes, with deep and mellow voices, often yield the best fremitus. Vocal fremitus is naturally felt better in thin-chested, lanky and hyposthenic individuals than in the thick-chested, fat and hypersthenic.

*Distribution of normal vocal fremitus* The intensity of fremitus varies greatly over different areas of the chest, even in health. It is normally felt somewhat better over the right side of the chest than over the left, because of the wider and shorter main bronchus on the right side, the asymmetrical position of the interbronchial septum, and the existence of an extra bronchus on the right side. Vocal vibrations are felt best below the right clavicle anteriorly, because of the nearness of the bronchial bifurcation, and posteriorly, between the scapulae, because of proximity of the larger bronchi. Vocal fremitus is usually less intense over the precordial region (because of encroachment of the heart on the lung) and over the scapula on either side.

ABNORMAL VOCAL FREMITUS Under pathological conditions, the tactile vocal fremitus may be either increased or decreased, or absent. It seems the only value of this sign is to distinguish dullness on percussion due to consolidation or pleural effusion.

Increase of vocal fremitus may be due to (1) Consolidation of lung, as in lobar pneumonia, caseous tuberculous pneumonia, pulmonary infarction or malignant disease, the increased fremitus being due to better conduction of voice sounds from the bronchi to the chest wall, through solidified lung tissue. (2) Collapse or atelectasis of lung with a patent bronchus, the bronchus being nearer to the chest wall because of shrinking of lung tissue, vocal fremitus is often better felt in this condition than over healthy lung tissue. (3) A superficial, thick-walled cavity in the lung, vocal fremitus is only increased when an area of consolidation surrounds the cavity.

Physiological increase of fremitus, associated with a deep voice or a thin and rigid chest wall must not be mistaken for the pathological fremitus of lung disease

Decrease or absence of vocal fremitus may be due to (1) disease of pleura such as pleural effusion, pneumothorax, haemothorax or thickened pleura, in case of effusion it is due to failure of conduction of vibrations through relaxed lung tissue (underneath the effusion) rather than through failure of conduction through fluid, (2) bronchial obstruction, (3) bronchial asthma or emphysema, (4) fibrosis of lung, or (5) thin-walled cavity within the lung

Physiological decrease of fremitus, associated with a high-pitched, shrill voice, or a thick or fatty chest wall, must not be mistaken for the pathological decrease of diseased states

Rhonchial fremitus In case of spasm of bronchioles or bronchi, as in bronchial asthma, emphysema or bronchitis, the air passing through the narrowed air-passages may produce vibrations or sounds called rhonchi. When palpable over the chest wall, these vibrations are referred to as rhonchial fremitus

Palpable rales Rales are moist sounds produced within the chest, as the result of air traversing through bronchi or bronchioles filled with secretion. When sufficiently loud, intense, gurgling, bubbling, low-pitched or coarse, rales may be palpable

Friction fremitus When pleural surfaces are roughened by inflammation, as in dry pleurisy, they give rise to friction sounds. When these sounds are accompanied by palpable vibrations over the chest wall, the phenomenon is described as a friction fremitus or palpable rub

Thoracic rigidity This is roughly measured by pressing the recumbent patient's sternum with the palm of the examiner's right hand, directly backwards, towards the spine. The resistance, encountered during the compression technique, is roughly proportional to the rigidity of the thorax. Increased rigidity may be due to senility, emphysema or pulmonary tuberculosis.

## PERCUSSION

Introduction The basic principles and technique of percussion have already been dealt with. The main purposes of respiratory percussion are (1) to determine the state of underlying tissues, such as lungs or pleura, from the degree of resonance of note elicited and sense of resistance encountered during

percussion (diagnostic percussion), and (2) to delineate or define the boundaries or borders of the lungs (topographical percussion)

Examination of the respiratory system by percussion demands considerable patience, training and ability to concentrate on (1) auditory perception of sound elicited, for intensity, character and pitch, and (2) tactile perception of the sense of resistance encountered. For best results, the examiner should familiarize himself with a combination of the two methods of auditory and tactile perception of the percussion stroke

#### METHODS OF PERCUSSION

Position of patient The patient may be percussed in the sitting, standing or recumbent position. The recumbent position is best avoided, except in the case of severely ill or moribund patients, who are too ill to sit or stand. The results obtained are unsatisfactory, owing to undependability of comparative percussion of the two sides of the back (the uppermost side being more resonant), and distortion of bodily contours by the underlying mattress. If the chest has to be percussed in the lying-down position, then the patient must be percussed in both right and left lateral positions

The standing position, although satisfactory from the point of view to results, is likely to prove tiring both to the patient and examiner

For routine examination, therefore, the sitting-up posture may be regarded as the position of choice. For percussion of the front wall of the chest, the patient sits on a stool opposite the examiner with the body bolt upright, completely relaxed, and with the sides symmetrical. For percussion of the back the patient bends slightly forwards with the head flexed on the chest, the shoulders sagging, and the arms resting, either crossed or uncrossed, on the thighs. During percussion of the interscapular and scapular regions, the patient is directed to place his hands over the shoulders, after crossing the arms in front of the chest. While the axillae are being percussed, the patient is instructed to put his hands over the head

Direction of percussion It is customary with most examiners to start percussion at the apices of the lungs, comparing identical or corresponding areas on the two sides, and slowly proceeding downwards, in similar manner, over the middle and lower zones of the lungs. In case of suspected apical tuberculosis, the procedure may be advantageously reversed, percussion being carried out from below upwards

The following procedure has served well in percussion of the lungs. Symmetrical areas on the two sides of the chest are compared, from above downwards, interspace by interspace, first along the anterior wall, then the axillae and finally along the posterior wall or back. Normal differences of percussion



note on the two sides, through the presence of the heart, stomach, and spleen on the left, and the liver on the right side, must be kept in mind whilst interpreting results

Percussion over the bones, such as the ribs and scapulae, is much less dependable than percussion along the interspaces or over the interscapular and infra-scapular regions

**Cardinal rules of technique** Correct respiratory percussion is by no means easy, requiring years of training and experience to perfect. In order to get the best out of percussion, the following rules must be rigidly observed

(1) The pleximeter, which is usually the middle finger (or index finger) of the examiner's left hand, must be firmly applied to the chest wall, so that no air-pockets are interposed between the finger and chest wall. The greater the thickness of the chest wall, the greater must be the pressure with which the pleximeter finger is apposed to the surface percussed. The other fingers of the examiner's left hand must be lifted away from the patient's chest in order not to vitiate results

(2) The plessor, which is usually the middle finger (or index finger) of the examiner's right hand, is kept flexed at a right angle and must hit the middle phalanx of the pleximeter finger, perpendicularly, with the pad and not the tip of the finger

(3) The percussion stroke must be sudden, the plessor finger being withdrawn immediately after the stroke, to prevent a damping of the note. The movement of percussion must originate at the wrist (which is kept completely relaxed) and not at the elbow or finger (except in case of gentle percussion)

(4) The force of the stroke must be varied according to the purpose of the percussion, the tissue or organ being percussed, the thickness of the chest wall, the area of the chest wall percussed, the age, sex and state of nutrition of the patient and the individual preference of the examiner. Heavy percussion is best avoided, as it is likely to involve an unduly large area of the lung at each stroke. Ideally, the force of the stroke should be just strong enough to elicit a clear and audible note. The force of the stroke must be kept absolutely constant when comparing symmetrical or other areas of the chest

(5) Percussion should proceed from resonant to dull areas or from "more resonant" to "less resonant" areas whenever possible, the auditory appreciation of any change of note being better in this direction

(6) When delineating the border of an organ, such as the heart or the liver, the long axis of the pleximeter finger must be kept parallel to the expected position of that border

(7) During percussion of the chest, it is better, whenever possible, to keep the pleximeter finger along an interspace rather than obliquely across ribs or interspaces, the latter may however prove necessary during percussion of the right and left cardiac borders

(8) The area percussed must be more or less equidistant from the two ears of the examiner, in order to prevent a wrong interpretation of sounds, the examiner must therefore directly face the centre of the patient's chest, whenever possible

Special techniques Besides the customary or classical method of percussion already described (employing a light, medium or heavy stroke), resort may be had, in special cases, to flicking percussion or "palpatory percussion"

Flicking percussion is a special form of light percussion, the surface to be percussed being flicked with the finger and thumb. It is useful for percussion of the abdomen, topographical percussion of cardiac borders and for eliciting metallic resonance in case of pneumothorax, whilst normally a flick on one side of the chest is heard as a dull thud or sound on the opposite side, in the case of pneumothorax the sound heard has a distinct metallic, ringing or chiming quality

Threshold percussion This is sometimes a useful method when the degree of resonance over the chest is in doubt. The area of suspected impairment is percussed progressively more lightly until a note is no longer obtained. The corresponding area over the opposite lung is then percussed with equal intensity. If a note is then obtained, it affords additional evidence of impairment upon the side originally suspected

Direct or indirect palpatory percussion, with a view to determining the sense of resistance (or vibration) to percussion, may be used to advantage for detecting the presence of fluid or consolidation within the chest. For this purpose, direct percussion with the pads of the three middle fingers of the right hand usually proves most effective, particularly when employed over the back of the chest

## TYPES OF PERCUSSION NOTES

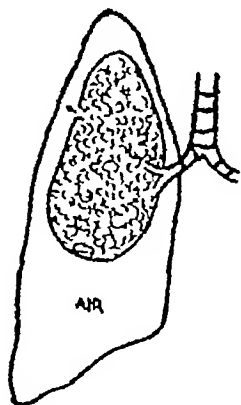
Normal lung resonance (Vesicular resonance) The normal percussion note of the chest is due to the underlying lung tissue, containing a normal amount of air in the air vesicles, air sacs and air passages. Usually referred to as normal lung resonance, the percussion note over healthy lung tissue has a distinctive and clear character with a low pitch. Since this note serves as the basis for comparison of various types of percussion notes, the examiner must familiarize himself with its distinctive characteristics and quality

It is important to realize, however, that normal lung resonance tends to vary slightly, not only from case to case, but over different areas of the chest in the same case. As a rule, the front wall of the chest yields a more resonant note than the back, because of the lesser bulk of musculature in front than at the back. Lesions that are more than 5 cm away from the chest wall, or smaller than 2 or 3 cm in diameter will not alter the percussion note

## ABNORMAL TYPES OF PERCUSSION NOTES

A large variety of percussion notes, different from normal lung resonance may be elicited on percussion of the chest wall, either in health or disease.

Abnormal types of percussion notes may be either (1) quantitatively different from normal lung resonance, as in case of tympany, subtympany, hyper-resonance, impairment of note, dullness and stony dullness, or (2) qualitatively different, as with cracked-pot resonance, amphoric resonance and bell tympany.

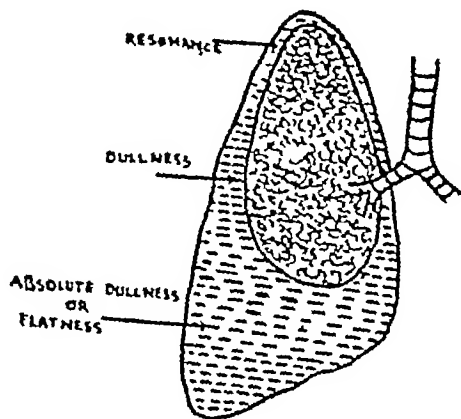


PNEUMOTHORAX  
Fig 10.24 Tympanic note on percussion  
Pneumothorax

**Tympany** (Tympanitic note) A drum-like note, elicited normally over the stomach intestine, trachea or larynx, is referred to as tympanitic note. When such a note is heard over a region of the chest wall, where vesicular resonance is normally encountered, the possibility of a superficial cavity in the lung or a pneumothorax (Fig 10.24) should be seriously considered. The so-called metallic tympany may be heard over an area of subcutaneous emphysema.

**Subtympany** (*Skodaic resonance*) (Fig 10.25) A hyper-resonant note, with a boxy quality, usually heard just above the level of a pleural effusion or pneumonic consolidation, results from the fluid or consolidation causing the lung to relax upwards and towards the hilum. The characteristic note over such relaxed lung tissue (partially filled with air) is described as subtympanitic or Skodaic.

**Hyper-resonance** (Hyper-resonant note) A note intermediate in pitch between normal lung resonance and tympany can be elicited, over *normal* lung tissue, by keeping the chest in full inspiration during percussion. Pathological hyper-resonance may be encountered either *bilaterally*, in case of emphysema (Fig 10.26 left), *unilaterally*, in case of pneumothorax or compensatory emphysema (Fig 10.26 right), or *locally* over one



PLEURAL EFFUSION

Fig 10.25 Zones of flatness and dullness, and band of skodaic resonance in pleural effusion

lung or lobe of lung, when the other lung or adjacent lung lobe is incapacitated by an extensive disease process

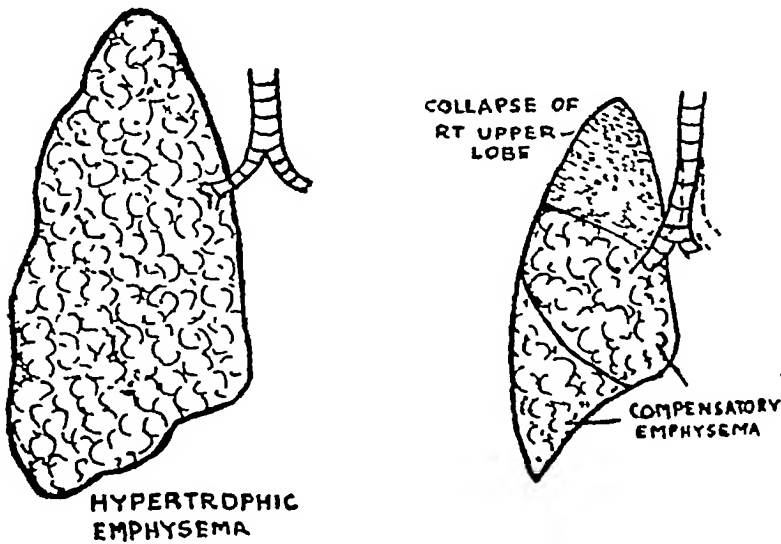


Fig 10.26 Hyper-resonance Emphysema (Left) Hypertrophic, (Right) Compensatory

Impaired note. When part of a lung becomes comparatively airless, as in consolidation (Fig 10 27), fibrosis or collapse, it fails to vibrate sufficiently to the percussion stroke, and gives rise to an impairment of note or slight loss of resonance. In such cases, the patch of consolidation or collapse of lung-tissue does not extend to the periphery of the lung, thus allowing some air to remain in the alveoli. It also may be heard over a patch of early tuberculous infiltration.

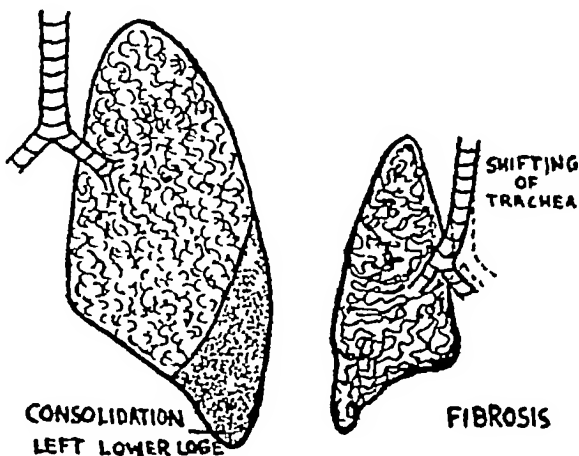


Fig 10.27 Impaired note (Left) Consolidation of lung (Right) Fibrosis of lung.

**Dull note.** An impairment of note, of greater degree than the one described above, is referred to as dullness. It may be encountered over consolidation of lung (as in lobar pneumonia, caseous pneumonia or carcinoma of lung), collapse of lung, thickened pleura, or extensive tuberculous infiltration of lung.

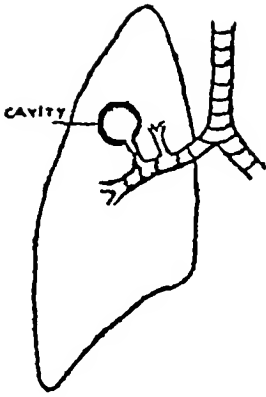
**Flatness (Absolute dullness)** (Fig 10 25) A percussion note, completely devoid of resonance or displaying absolute or extreme dullness, is referred to as a flat or stony dull note. It can be elicited normally by percussing the thigh of the patient. A flat note is classically encountered over a pleural effusion (volume of free fluid should be more than 200 ml), or empyema, the fluid in such a case being able to damp down the vibrations of both chest wall and underlying retracted lung. The percussion note may also be flat over a solid intrathoracic tumour, or lung fibrosis associated with pleural thickening.

**Stony dullness** Though it is customary to refer to most marked degrees of dullness as in pleural effusion as stony dullness, the correct definition would be a type of absolutely dull percussion note associated with pain when percussing in the examiner's pleximeter finger as one would experience when percussing over a stone. This may be met with when percussing over a large solid tumour in the lung such as carcinoma.

Type of percussion note	Causative lesion
Tympanic	Gas containing hollow viscera
Subtympany (boxy note)	Above pleural effusion or consolidation
Hyperresonant	Pneumothorax
Resonant (Normal)	Normal aerated lung
Impaired	Pulmonary fibrosis, sometimes consolidation or collapse
Dull	Pulmonary consolidation or collapse (some cases) Thickened pleura, tumour, raised diaphragm
Flat	Pleural effusion
Stony dull	Massive growth in lung or pleura

#### Other abnormal percussion notes

**Cracked-pot resonance** This is a special variety of tympanic resonance, which can be elicited normally over the chest of an infant or child during the act of crying. Pathologically, it is found over a lung cavity that is in communication with a bronchus (Fig 10 28). It is due to the sudden expulsion of air from the cavity into the bronchus, through the narrow opening or communi-



CAVITY

Fig 10.28 Cracked-pot resonance Lung cavity in communication with bronchus

cation The cracked-pot note can be artificially imitated by clasp[ing] the hands loosely together and then striking the clasped hands against the knee

Amphoric resonance A low-pitched and hollow note, that can be artificially reproduced by percussing the normal trachea or one or other cheek (moderately distended with air), may be encountered over a pneumothorax or large cavity in the lung

Bell tympany This is a high-pitched, tympanic or metallic sound, heard over the chest in case of massive pneumothorax When a silver coin is placed flat on the affected side and percussed with a second coin, the ear or stethoscope, when applied directly over the opposite side of

the chest may detect a clear bell-like sound variously referred to as the coin bell sound or brass sound, sound (bruit d'airain) It has been likened to the "chiming of a church bell" or the "sound of hammer on an anvil"

#### TOPOGRAPHIC PERCUSSION OF LUNGS

Percussion of the chest, when usefully employed to determine the boundaries or extent of lungs, is referred to as topographic percussion This is of particular value in determining the upper or apical border of the lung (apical percussion), the lower border (basal percussion), and the anterior border of the left lung (which corresponds, in fact, to the outer and upper borders of the area of superficial cardiac dullness) The range of excursion or expansion of the lung either at the base or apex can be determined by percussing either the basal or the upper border of the lung during deep inspiration and expiration (tidal percussion)

Apical percussion Percussion of the apical regions of the lungs should be undertaken routinely, during physical examination of the chest, as it may afford information of diagnostic value, particularly in case of early pulmonary tuberculosis The percussion should be comparative (comparing the extent and nature of lung resonance at the two apices) and must employ a light stroke

Apical percussion can be carried out quite simply, in the supraclavicular fossae anteriorly, by determining the upper borders of lung resonance on the two sides Normally, the upper border is from 3 to 5 cms above the clavicle, on either side, being at times somewhat higher on the right side Diminution or absence of the supraclavicular zone of resonance (on one or both sides) is good evidence of pulmonary tuberculosis (unilateral or bilateral) On the other hand, an increased extent of resonance, bilaterally, suggests emphysema

An alternative method of percussion of the apical regions of the lungs depends on mapping *Kronig's isthmus* on either side. This is an area of band resonance, over each shoulder, connecting the large zones of lung resonance over the anterior and posterior aspects of each side of the chest. *Kronig's band of resonance*, which corresponds roughly to the apex of the lung, is usually 5 to 7 cms in width (two-finger-breadths on the right side), and is bounded medially by a "dull" zone, corresponding to the structures of the neck, and laterally by the "dullness" of the shoulder region. Whilst diminution or absence of *Kronig's isthmus*, on one or both sides, is suggestive of apical tuberculosis (unilateral or bilateral), an increase in the width of the isthmus on both sides is suggestive of emphysema. The band of resonance can be mapped out, quite simply, by percussing medially or inwards from each acromioclavicular joint, along a straight line across the shoulder.

In special cases, the entire upper or apical border of the lung can be delineated from back to front, by percussing along various vertical lines. Such an investigation proves unnecessary for most cases. In recent years, specialized percussion of lung apices and mapping out of *Kronig's bands of resonance* have been more or less entirely replaced by radiographic methods of diagnosis.

**Basal percussion** Normally, during quiet breathing, the lower border of lung resonance corresponds to the *sixth* rib in the midclavicular line, *eighth* rib in the midaxillary line and *tenth* rib in the scapular line, it may be at a slightly lower level on the left than on the right side. The lower border tends to be somewhat higher in children and lower in elderly individuals than in adults.

Percussion of the lower border of the lung necessitates light percussion anteriorly and heavy percussion at the back (because of the thicker musculature). A change of note from vesicular resonance to dullness (posteriorly, on both sides, and anteriorly, on the right side) or from vesicular resonance to tympany (anteriorly, on the left side because of Traube's area of tympany), when percussing downwards over the chest, serves to delineate the lower or basal limit of lung resonance.

The lower border of the lung resonance tends to be *depressed* in case of emphysema or pneumothorax, and *raised* in case of lung fibrosis, collapsed lung, consolidation, ascites, massive abdominal tumour or pleural effusion.

**Anterior border of left lung** Corresponding in extent to the upper and outer borders of the area of superficial cardiac dullness, the anterior border of the left lung can be mapped out by percussing upwards and outwards, from the inner end of the fourth left interspace, in various directions, until the dull note of cardiac dullness is replaced by the resonant note of normal lung tissue.

**Tidal percussion (Philip's method)** Percussion of the lower border of lung resonance, on each side, at the height of deep inspiration and expiration, serves to determine the degree of range of respiratory expansion or movement of the lung. Referred to as "tidal percussion", this procedure frequently affords diagnostic information. Restriction or diminution of movement of the lower border of lung resonance, either unilateral or bilateral, during respiration, is suggestive of some disease of the lung, such as pulmonary fibrosis. The degree of movement of the apical or upper border of the lung, during the two phases of respiration, can be similarly determined by percussion over the supraclavicular fossae, unilateral restriction of movement at the apex is highly suggestive of tuberculous infiltration.

## AREAS OF ABNORMAL PERCUSSION NOTE IN HEALTH

During percussion of the normal healthy chest, one encounters certain areas over which the note elicited fails to conform to the classical description of normal lung resonance, the most important of these being

*Area of cardiac dullness* On the left side of the chest anteriorly there is an area of dullness extending in triangular fashion from the sternum (on the right) to the junction of the midclavicular line with the fifth left interspace. The percussional dullness of this area is caused by the subjacent heart.

*Area of liver dullness* Over the lower part of the right side of the chest, anteriorly and laterally, one can map out an extensive area of dullness, corresponding in position to and caused by the underlying liver.

*Area of splenic dullness* A smaller area of dullness or diminished resonance, in the 8th interspace in the midaxillary line, is similarly attributable to the presence of the spleen on that side.

*Traube's area* (Traube's semilunar space) At the lower border of the left lung anteriorly, pulmonary resonance is replaced by a drum-like, tympanitic note over a semilunar area (Traube's area), dependent on the presence of the stomach. This area is bounded above by pulmonary resonance, on the right by liver dullness, on the left by splenic dullness and below by the left costal margin. Its size is dependent on the contents of the stomach.

## SPECIAL PERCUSSIONAL FINDINGS IN DISEASE

Reduction of both cardiac and liver dullness occurs in hyperinflation of the lungs

*S-shaped curve of Ellis* In moderate-sized effusions within the pleural sac, the upper border of dullness or flatness, which is highest in the axilla and lowest at the spine, tends to assume the shape of the letter S, and hence is referred to as the S-shaped curve. The exact nature of the phenomenon remains obscure. Attributed by some to "capillary suction", a probable mechanism suggested is that with a moderate effusion, the lung tends to retract towards the hilum. The part of the lung below the hilum being fixed by the pulmonary ligament, the fluid tends to rise in the axilla, as in this region the lung is more free to collapse.

*Grocco's triangle* (Paravertebral triangle of dullness, Grocco's sign) An area of relative dullness or impairment of note, roughly triangular in shape, can sometimes be mapped out by percussion, in case of a large or medium-sized pleural effusion, over the back of the chest, on the contralateral side or side opposite to the effusion. The triangle is bounded medially by the midspinal line from above the level of the effusion to the level of the tenth dorsal vertebra (which corresponds to the level of the lower or inferior border of normal lung resonance), below by a horizontal line extending outwards from the tenth dorsal vertebra along the lower limit of lung resonance for a distance of about 3 to 7 cm, and laterally by a somewhat curved line connecting these two lines. The presence of Grocco's sign is as a rule evidence of pleural effusion.

The classical explanation for Grocco's sign is that it is due to a lateral shift of the posterior mediastinum to the opposite side as the result of fluid. According to an alternative explanation, fluid interferes with the vibrations of the chest wall not only on the side of the effusion, but also, to a less extent, on the opposite side. The triangular shape of the area of dullness may be due to the lung being wedge-shaped at the base and to its increasing thickness laterally and upwards.



**Garland's triangle** In the case of a moderate-sized or large pleural effusion, the lung on the side of the effusion floats upwards and backwards, its lower part being "relaxed". The roughly triangular area, with a slightly tympanic note or Skodaic resonance that may be elicited in such a case by percussion over the relaxed area of lung is referred to as Garland's triangle. It is an inconstant sign of little or no value.

**Obliteration or encroachment of Traube's area** Traube's semilunar area of tympany, over the anterior and lower aspect of the left side of the chest, may be partially obliterated from above by a zone of dullness, in case of a large left-sided pleural effusion. This is due to filling up of the complementary pleural space or lower part of the pleural sac by the fluid and depression of the diaphragm. The band of flatness due to fluid is interposed between the lung resonance above and stomach tympany below.

**William's tracheal resonance** An area of tympany over the first or second interspace, close to the sternum, may be due to a patch of consolidation or fibrosis interposed between the trachea or a major bronchus and the chest wall. It may be wrongly attributed to a cavity in the lung. This sign is referred to as the "pulled trachea syndrome" in case of fibrotic apical tuberculosis.

**Wintrich's sign** When the percussion note over an area of the chest wall, during inspiration, appears clearer and higher pitched with the mouth open than with it closed, it is referred to as "Wintrich's change of tone". It may be due to a lung cavity communicating with a bronchus, a pneumothorax or mediastinal tumour.

**Gerhardt's sign** When the percussion note over an area of the chest wall appears lower-pitched with the patient recumbent than with him standing or sitting, it is referred to as "Gerhardt's change of tone". It is usually due to a lung cavity containing both fluid and air, the shape of the cavity altering with change of posture.

**Friedreich's sign** When the percussion note, over an area of the chest wall, becomes higher in pitch during forced inspiration than during expiration, it is suggestive of a lung cavity. The change of note depends on an increased amount of air during inspiration and alteration in tension of the walls of the cavity.

**Movable or shifting dullness** In the case of hydro- or pyo-pneumothorax, the upper border of dullness or flatness is sharply defined and remains horizontal, irrespective of the position of the patient. It is possible to demonstrate, in such cases, a shift or change in the position of the fluid with change in posture of the patient. The upper border of dullness is delineated by percussing downwards, over the anterior chest wall, with the patient upright, the pleximeter finger is then kept in place over the dull area and the patient made to lie down, re-percussion over the same (previously dull) area, after a few seconds to allow the fluid to fall away from the chest wall, will now reveal it to be resonant. The degree of "shift" can be measured by percussing downwards in the new position, until a dull note is re-obtained. A shifting dullness may also be found in case of a moderate-sized pleural effusion, but never with an interlobar or loculated effusion.

**Lines of demarcation** In case of an area of dullness being discovered during routine percussion of the chest, an attempt must be made to determine its extent by percussing lines of demarcation between it and the surrounding areas of vesicular resonance. This may help to determine the true nature of the causative lesion. Whilst the extent of dullness in lobar pneumonia corresponds to the anatomical limits of a lung lobe, and the dullness of a cyst, aneurysm or interlobar effusion is usually sharply defined or demarcated from surrounding areas of resonance, the area of impairment in case of pulmonary tuberculosis or new growth usually fails to display a clear outline or line of demarcation.

**Myotatic irritability** In states of emaciation or wasting, as in cases of pulmonary tuberculosis, a percussion stroke over the front of the chest, close to the sternum, may cause the transitory appearance of a localized swelling (myoidema) or a transient twitching or fibrillary

contraction (myotatic irritability) in a neighbouring muscle. Usually indicative of excessive irritability of muscle tissue, this sign may be more marked or confined to the side of the pulmonary affection

## AUSCULTATION ✓

As in the case of the cardiovascular system, auscultation of the lungs yields more information than any of the other three classical methods of physical examination.

Methods of auscultation Auscultation of the lungs can be carried out either with the unaided ear applied to the chest, or with the aid of a stethoscope. Except for detection of high-pitched or blowing sounds of low intensity, such as the tubular breathing of lobar pneumonia, and for bone-conducted sounds, the indirect method using a binaural or standard stethoscope is as a rule adopted routinely in preference to the direct method. For exact localization of sounds, comparison of symmetrical areas and auscultation of areas inaccessible to the unaided ear (such as the pit of the axilla or the supraclavicular fossa), the indirect method is unquestionably the method of choice.

Position of patient. For auscultation of the lungs, the ideal posture or attitude for the patient is upright, either *sitting* or *standing*, for examination of the back, the patient may lean slightly forwards, with the head flexed and arms crossed in front or resting on the thighs. The patient and examiner must be both mentally and physically relaxed and comfortably placed at the time of examination.

Examination in the *recumbent* or lying down position, although undesirable, may prove obligatory in the case of a seriously ill or *bed-ridden* patient. In such a case, the patient's back must be auscultated first with him lying on one side and then on the other. In case turning in bed becomes inadvisable, as in the case of moribund patients, the Bowles type of chestpiece may be gently slipped underneath the patient, for listening to the back.

Method of breathing Since normal or quiet breathing usually proves inadequate for a proper study of auscultatory sounds, resort must be had to *forced* or *deep* breathing, through the mouth, in all cases. The correct manner of breathing must be clearly explained or demonstrated to the patient prior to auscultation. Defective auscultation of respiratory sounds may be due to the patient breathing (1) through the nose, especially in the presence of nasal obstruction, (2) noisily or too forcibly, thus causing the production of adventitious sounds from the mouth, or (3) in a shallow or incorrect manner, mainly because of pain on breathing.

If the patient fails to co-operate or follow instructions about the correct method of breathing, he may be asked to count numbers or give a few successive coughs, the deep inspiration, following on such a procedure, being closely listened to by the examiner with a stethoscope. This method is however likely to exhaust both patient and examiner.

## SCHEME OF EXAMINATION

It is necessary to interview gale systematically the following features during auscultation of the respiratory system: (1) character and type of breath sounds over symmetrical areas of the chest (2) presence or absence of adventitious or added sounds (adventitious) and (3) character of the vocal resonance inclusive of both spoken and whispered voice.

## NORMAL BREATH SOUNDS

In order to eliminate an auscultation finding, it is essential for the examiner to familiarize himself thoroughly with the respiratory sounds of health; without a proper conception of normal standards, he cannot possibly hope to interpret abnormal auscultatory findings.

**FEATURES TO NOTE.** The respiratory murmur is a composite sound, usually made up of two elements the inspiratory sound and the expiratory sound with or without an intermediate pause or interval. The following features must be observed in the case of the breath sounds: (1) intensity or loudness, (2) quality or character, whether rustling, breezy, blowing or tubular, (3) comparison of inspiratory and expiratory elements of the sound from the points of view of intensity, duration or length and pitch, (4) presence or absence of intermediate pause, between inspiration and expiration, (5) other characteristics such as prolongation, or jerky or interrupted nature, (6) presence of other sounds or accompaniments.

**Mode of production:** Normal breath sounds are generated by turbulent airflow in upper airways i.e. in the pharynx and larger airways of the lungs (frequency range of 200 to 2,000 Hertz or cycles per second). The characteristic rustling sound is heard with the stethoscope over the upper sternum. This is bronchial cretling, normal at this site.

As this sound is transmitted through the lungs it is damped, the higher frequencies are lost and a softer, lower pitched sound (200 to 400 Hz) is heard. These are normal breath sounds heard at the lung base. In the smaller airways air flow is slower and laminar, turbulence and sound cannot develop (hence the term vascular breathing is not quite correct). The smaller airways and alveoli are therefore a filter and not a source of lung sounds.

**FACTORS DETERMINING TRANSMISSION OF BREATH SOUNDS.** The intensity of the breath sounds heard through the chest wall depends on (a) rate of airflow into the territory of the lung under the stethoscope, (b) the acoustic properties of the two media namely the lung and the chest wall. Transmission is almost complete between two well matched media like consolidated lung and the chest wall, hence the similarity between bronchial breathing and the breath sounds heard directly over the trachea. On the other hand, sound is

reflected at the interface between the lung and air or fluid in the pleural cavity so that in pneumothorax and in pleural effusion no breath sound reaches the chest wall

Types of normal breath sounds The following types or varieties of breath sounds are normally audible over different parts of the healthy chest (1) vesicular breathing, over most areas of the chest, (2) tracheal or tracheo-bronchial breathing, over the larynx, trachea and lower cervical spine, and (3) bronchovesicular or mixed breathing, over and around the upper part of the sternum and third and fourth dorsal vertebrae

GRAPHIC RECORDING A certain code of symbols has been found convenient in practice for the diagrammatic representation of various types of breath sounds (Figs 10 29, 10 30) Normally, the upstroke of the symbol represents its inspiratory element, and the downstroke its expiratory element Whilst the length of each stroke corresponds to its duration or timing, and the thickness to its loudness or intensity, the angle between the upstroke and downstroke represents the pitch of the respiratory sound

Vesicular breath sounds The type of breathing heard over normally functioning lung tissue is referred to as vesicular breathing

CHARACTERISTICS The main characteristics of vesicular breath sounds are (1) quality of sound, which is characteristically "rustling" or "breezy", not unlike the "rustle of tree-leaves in the wind", (2) relatively greater intensity of the inspiratory sound, the same being much louder than the expiratory sound, (3) longer duration of the inspiratory sound, being three to five times as long as the expiratory sound, (4) the lower pitch of the expiratory sound compared to that of the inspiratory, and absence of a pause or interval between inspiratory and expiratory sounds Attention to these features usually facilitates the recognition and differentiation of vesicular from other varieties of respiratory sounds

It may be noted, however, that the rustling or breezy quality of vesicular breathing is mainly a subjective impression on the part of the examiner, whilst a pause between inspiratory and expiratory sounds may be noted in patients who tend to "hold the breath" for a second or two after each inspiration

DISTRIBUTION Vesicular breathing, which is typical of normally aerated lung parenchyma, is heard all over the chest in health, except in the following areas (1) over the larynx, trachea and lower cervical vertebrae, and (2) over and around the upper part of the sternum and third and fourth dorsal vertebrae

INTENSITY The loudness or intensity of vesicular breathing is not uniform all over the chest It tends to be particularly intense and characteristic in the axillary and infrascapular regions, and over the upper part of the front wall

of the chest. Near the lower margins of the lungs and over the scapular regions, the vesicular breath sounds are normally somewhat "distant", "diminished" or "quiet."

**MODE OF PRODUCTION** The classical view, since the time of Laennec, has been to attribute the vesicular component of the respiratory sound to distension and separation of the alveolar walls by the in-rushing current of air. According to another school of thought, the laryngeal sound, whilst travelling down the bronchi into the alveoli, gets modified through the addition of other sounds (produced by the passage of air from narrow terminal bronchioles into the wide vestibules) and acquires a rustling or vesicular quality.

Although the actual duration of expiration is slightly longer than that of inspiration (ratio of 6 to 5), its auditory duration is much shorter than that of inspiration (ratio of 1 to 3 or 1 to 4), this is due to the elastic recoil of the distended lungs being maximal at the onset of expiration, resulting in a short and low-pitched expiratory sound.

**Tracheal or bronchial breath sounds.** By tracheal breathing is meant the respiratory sound that is heard, in health, over larger air passages, such as the larynx and trachea. Although similar to the bronchial breathing of diseased states and at times wrongly referred to as bronchial breathing, tracheal breathing is usually louder than true bronchial breathing, being produced in larger air passages

**CHARACTERISTICS** The main characteristics of tracheal breathing (as in case of bronchial breathing) are (1) a characteristically "blowing", "hollow" or "tubular" quality, (2) an expiratory sound somewhat longer in duration than the inspiratory, (3) a distinct pause or interval between inspiratory and expiratory sounds, and (4) an expiratory sound higher in pitch and louder in intensity than the inspiratory

Tracheal (or bronchial) breathing can be differentiated from vesicular breathing by its (1) characteristic blowing quality, (2) greater intensity, (3) long duration of the expiratory sound, (4) higher pitch of both sounds, and (5) presence of a pause between inspiration and expiration.

**DISTRIBUTION.** Tracheal breathing is normally audible over the larynx, trachea and lower cervical vertebrae.

**MODE OF PRODUCTION** Tracheal breath sounds are due to the in and out movement of air through the narrow aperture of the glottis. The lower pitch of the sound during inspiration is due to the glottic aperture being wider during inspiration, because of a wider separation of the vocal cords

**Bronchovesicular breath sounds.** A type of breathing, intermediate in character between vesicular and tracheal (or bronchial) breathing, is referred to as bronchovesicular (indeterminate or mixed) breathing. Depending on

the relative preponderance of the "vesicular" and "bronchial" elements, an attempt has been made, by some, to distinguish two varieties of indeterminate breath sounds, viz, vesiculobronchial and bronchovesicular, a distinction that is both difficult and unnecessary

**CHARACTERISTICS** In view of its composite nature, and the varying preponderance of one or other element (vesicular or bronchial), the clinical characteristics of bronchovesicular breathing are somewhat ill-defined and variable. Its recognition usually depends on the nature of the expiratory sound, when the latter is louder, longer, and higher in pitch than the inspiratory sound, or displays a "hollow" character (in an otherwise typical instance of vesicular breathing), the possibility of bronchovesicular breathing should be considered

**DISTRIBUTION** Bronchovesicular breathing is normally heard over and around the upper part of the sternum, near the third and fourth dorsal spines between the scapulae, and at times over the lung apices, particularly on the right side

**MODE OF PRODUCTION** Bronchovesicular breathing usually arises when normal air-containing lung tissue is interposed between a large bronchus and the chest wall, thus combining the characteristics of both vesicular and bronchial types of breathing

## ABNORMAL BREATH SOUNDS

**TYPES OF ABNORMAL BREATH SOUNDS** The most important varieties of abnormal respiratory sounds are (1) abnormal vesicular breath sounds, (2) bronchial breath sounds, and (3) bronchovesicular breath sounds. Unusual varieties of abnormal breath sounds such as metamorphosing and asthmatic types of breath sounds, may also be heard in disease

**MODE OF PRODUCTION** Abnormal breath sounds are heard if they are abnormally generated and if they are abnormally conducted

(a) Abnormal generation of breath sounds occurs mainly in presence of narrow airways due to intensification of linear velocity within them, so increasing turbulence and thus the breath sounds become louder. In most patients with chronic bronchitis or asthma, loud breath sounds can be heard at the mouth without a stethoscope, or even at a distance

(b) Abnormal conduction Abnormal lung conducts the centrally generated breath sounds abnormally. The consolidated lung of pneumonia is a more efficient sound conductor than normal lung, whereas the overinflated lung of emphysema is a less efficient sound conductor. The liquid surface of an effusion where it meets the lung reflects sound away from the listening stethoscope and so the intensity of breath sounds is reduced

**Abnormal types of vesicular breathing** The following varieties of abnormal vesicular breath sounds are recognizable in disease (Fig 10 30) (1) exaggera-

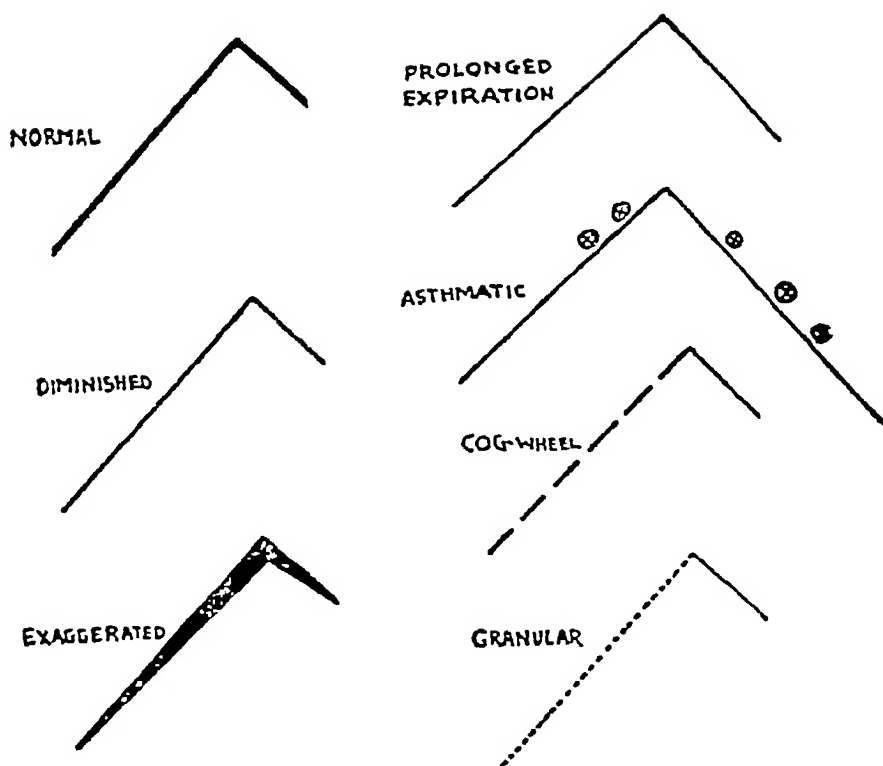


Fig 10.29 Graphic representation of vesicular breath sounds. Asthmatic breath sounds accompanied by rhonchi are also shown.

ted (harsh and puerile) breath sounds, (2) diminished or feeble breath sounds, (3) absence of breath sounds, (4) vesicular sounds, with prolonged expiration, (5) asthmatic breath sounds, (6) cogwheel (interrupted or jerky) type of breathing, and (7) granular breathing of Grancher. Abnormalities of vesicular breathing are usually dependent on alterations of intensity, duration or continuity of one or both phases of the respiratory murmur.

Exaggerated or loud breath sounds. Although vesicular in character, the breath sounds show an increase of intensity or harshness, an abnormality that may be either physiological or pathological. In children, thin-chested individuals, and during states of excitement, overexertion or overbreathing, the breath sounds may appear harsh or exaggerated. In women, the breath sounds are frequently louder than in men because of the costal or thoracic type of breathing.

Pathologically, harsh, puerile or exaggerated breathing may be noted bilaterally in states of dyspnoea or bronchitis, and unilaterally, or regionally in case of pulmonary tuberculosis or compensatory emphysema.

In the event of a lung (or part of a lung) being rendered functionless by disease as in case of pleural effusion, pneumothorax, severe kyphoscoliosis, bronchial obstruction, malignant disease or consolidation, the healthy lung (or healthy part of the affected lung) tends to become emphysematous (compensatory emphysema) and displays exaggerated vesicular breath sounds (compensatory or complementary breathing)

At rest the inspiratory breath sounds in a healthy subject are inaudible at the mouth. As mentioned earlier the source of much of the sound audible during breathing is the upper respiratory tract and the first few generations of bronchi. In narrow airways the high velocity intensifies these irregular flow patterns and the breath sounds become louder. The noise of inspiration at the mouth heard by the unaided ear is louder, greater the obstruction and correlates well with the forced expiratory volume in one second (FEV<sub>1</sub>) and other indices of airflow obstruction. But in patients with emphysema the inspiratory noise is less loud than might be expected from the decrease in FEV<sub>1</sub>. An audible or faint inspiratory sound at the mouth in the presence of a low FEV<sub>1</sub> helps to diagnose primary emphysema (where the inspiratory calibre of the large bronchi is normal) from other obstructive diseases of the airways. In focal stenosis caused by a tumor, scarring or a foreign body partly obstructing the trachea or one of the principal bronchi, the inspiratory breath sounds are louder than predicted by FEV<sub>1</sub>.

Diminished or feeble breath sounds Although retaining its vesicular character, the intensity of breath sounds may be reduced owing to loss or diminution of intensity. This may be physiological in habitual shallow-breathers and during quiet breathing, the respiratory murmur can be brought out in such cases by resorting to forced breathing. Distant breath sounds may be associated with a thick chest wall or obesity. Pathological diminution of breath sounds may be due to defective production or defective conduction of the respiratory murmur. Defective production of respiratory sounds may result from (1) chest pain, as in case of dry pleurisy, pericarditis or painful affection of the spine, (2) weakness or paralysis of respiratory muscles, as in acute anterior poliomyelitis, (3) diminished excursion of the diaphragm as in massive ascites or intra-abdominal tumour, (4) loss of elasticity of lung tissue, as in emphysema or lung fibrosis, (5) decreased alveolar capacity, as in pulmonary oedema or early stage of lobar pneumonia, or (6) defective flow of air through the air passages, as in bronchitis, oedema of glottis or occlusion of the trachea or bronchus. Defective conduction of the respiratory murmur may result from (1) affections of the pleura, such as pleural effusion, hydrothorax, pneumothorax, thickened pleura or malignancy of pleura, or (2) pathological thickening of chest wall, as in malignant infiltration.

Of the numerous causes of feeble breath sounds mentioned above, the commonest is pleural effusion. Pleural fluid, whether of the nature of an exudate or transudate, is a good conductor of sound and hence not respon-



sible for the non-conduction of breath sounds. It is actually the "relaxed" lung tissue, underneath the effusion, that is responsible for failure of transmission of the respiratory murmur. Vesicular breathing, although diminished or absent in two of every three cases of pleural effusion, may be, paradoxically enough, bronchial or of tubular type in the remainder.

*Absence of breath sounds* Normal respiratory sounds may be completely absent or "missing" in case of a large pleural effusion, hydrothorax, pneumothorax, or area of lung collapse, secondary to complete occlusion of a bronchus. Absence or diminution of the vesicular murmur, in such a case, depends on (1) the degree of interference to normal lung ventilation or aeration by the causative lesion, and (2) the extent or thickness of the non-conducting medium, interposed between the seat of production of the respiratory murmur and the area of chest wall auscultated.

Although the loudness of breath sounds heard through the chest wall depends on the rate of airflow into the lung territory under the stethoscope, the acoustic properties of the lung and chest wall are equally important. Impaired air entry should not be used as a synonym for poorly audible breath sounds.

*Prolonged expiration* Normally, the duration of the expiratory sound is about a third of that of the inspiratory sound. Although typical of bronchial or bronchovesicular breathing, prolongation of the expiratory sound may also occur in association with vesicular breathing in case of early tuberculous infiltration (usually, unilateral and localized), bronchitis, asthma or emphysema. Whilst in pulmonary tuberculosis prolongation of expiration is due to local loss of elasticity of lung tissue, in asthma, bronchitis and emphysema it is due to a slowing down of the expiratory act by the increase of resistance offered to the egress of air from the alveoli by the narrowed bronchioles.

*Asthmatic breathing* In bronchial asthma, with or without associated emphysema or bronchitis, the breath sounds tend to be characteristic. The expiratory sound becomes harsh, markedly prolonged and wheezy in character, whilst the inspiratory sound remains normal or short, and is associated with rhonchi and rales.

*Cog-wheel breathing (Interrupted or jerky breathing)* (Fig. 10.30) When the continuity of the inspiratory element of the respiratory murmur is broken, interrupted or jerky, the condition is referred to as *cog-wheel breathing*. It may be observed, physiologically, in highly nervous or neurotic individuals and during nasal breathing in subjects of chronic nasal obstruction. Pathologically, cog wheel breathing, over the apical region, may be associated with early pulmonary tuberculosis. The pathological variety of interrupted breathing, unlike the physiological, tends to persist even during forced breathing. Cog-wheel inspiration is no longer considered a physical sign of importance.

The jerky or interrupted nature of cog-wheel breathing may be due to (1) unequal or arrhythmic expansion of alveoli, through loss of elasticity of local patches of lung tissue, (2) involvement of smaller bronchioles, interfering with the free ingress of air into the alveoli, or (3) small pleural adhesions.

*Granular breathing* This is actually a finer variety of interrupted breathing with the number of pauses or jerks during inspiration greatly increased. It has been regarded by some as an early sign of tuberculous infiltration of the lung.

*Bag pipe sign* With partial bronchial obstruction, the positive pressure in the lung persists for an appreciable interval after the expiratory effort is stopped, and the outflow of air continues and can be heard on auscultation.

**Bronchial breathing.** Bronchial or tubular breathing, although closely similar to the "tracheal breathing" heard normally over the larger air passages, such as the trachea and larynx, differs from the latter in being less intense and harsh but of a higher pitch, this is because of its being produced within smaller or narrower air passages.

True bronchial breathing, when heard anywhere over the chest, is always indicative of disease, and must therefore be looked for carefully and viewed with concern whenever present.

**CHARACTERISTICS** The characteristic features of bronchial breath sounds must be memorized: (1) a high-pitched inspiratory sound, (2) a long intermediate pause between inspiration and expiration, (3) a very harsh and high-pitched expiratory sound, (4) marked prolongation of expiration (the latter being equal in length or longer than inspiration), and (5) a distinctive character or quality, variously described as "blowing", "tubular", "hollow", "aspirate" or "guttural" and crudely imitable by whispering the syllable "ha".

Bronchial breathing must be clearly differentiated from normal vesicular breathing, because of its pathological implications by attention to the following points:

<i>Vesicular breath sounds</i>	<i>Bronchial breath sounds</i>
Soft low pitched and rustling in type	Loud, higher in pitch and blowing
Inspiratory phase audible throughout and longer than expiratory phase or silent phase	Pitch of expiratory sound higher and duration longer than that of inspiration
Pause between inspiratory and expiratory sounds	No pause between inspiration and expiration

**TYPES** Depending on the site, extent and nature of the causative lesion and its relationship to adjacent air passages, bronchial breathing may show variations of intensity and pitch. It may be either loud (exaggerated), moderately intense, or soft (diminished) depending on loudness or intensity, and *low-pitched (cavernous)*, *medium-pitched*, or *high-pitched (tubular)*, depending on the pitch of the sounds (Fig 10 31). When associated with a distinctive "echo-like" reverberating quality, bronchial breathing is referred to as *amphoric breathing*.

**MODE OF PRODUCTION** Normally, the laryngotracheal sound is modified by the air cells of normally functioning lung tissue into the characteristically "rustling" vesicular murmur. When normally aerated lung tissue is replaced by consolidation of lung, the glottic sound is conducted to the chest wall without modification through the solidified lung tissue. On the size or capacity

of the underlying air passage or bronchus (whether large, medium or small) depends the nature or pitch of the bronchial breath sound, whether cavernous, medium-pitched or tubular. A lung cavity being somewhat of the nature of a large-sized bronchus usually gives rise to cavernous or low-pitched bronchial breathing.

**SIGNIFICANCE** Cavernous or low-pitched bronchial breathing, with its peculiar "hollow" character, can be normally heard with a stethoscope over the occipital region of the skull. It can be artificially imitated by breathing into the hollow of one's hand or by whispering the syllable "who". When heard over the chest, cavernous breathing is sug-

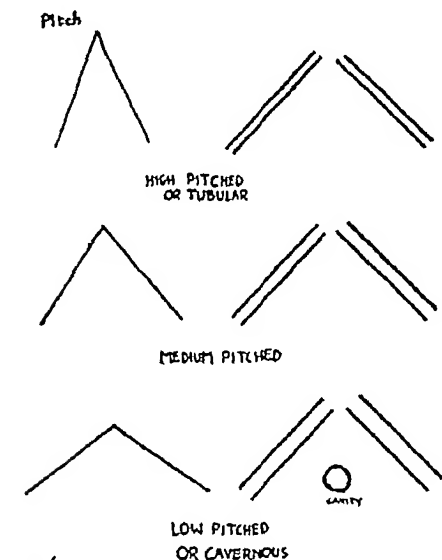


Fig 10.30 Graphic representation of bronchial breath sounds

gestive of (1) an underlying cavity in the lung (with uneven or irregular walls), (2) an open pneumothorax, or (3) a trachea deviated to one or other side by apical fibrosis of the lung, usually secondary to tuberculosis ("pulled trachea syndrome") A deviated trachea may therefore lead to an erroneous diagnosis of cavitation at the lung apex.

Tubular or high-pitched bronchial breathing, with its characteristic "tubular" or "aspirate" quality, whenever present, is suggestive of consolidation of lung tissue, overlying small-sized bronchial tubes. It may therefore be encountered in cases of lobar pneumonia (where tubular breathing is seen at its best), caseous or tuberculous pneumonia, pulmonary infarction, atelectasis or collapse of lung (secondary to compression or partial obstruction of a bronchial tube), malignant disease or massive tuberculous infiltration of lung. Solidification of lung, whether resulting from inflammation as in lobar pneumonia, engorgement of blood as in pulmonary infarction or collapse of air vesicles as in

atelectasis, gives rise to bronchial breathing provided some adjacent bronchus or bronchi remain partially open or patent

Another cause of high-pitched bronchial breathing is, paradoxically enough, massive pleural effusion. In about a third of all cases of massive pleural effusion or empyema (especially post-pneumonic), the breath sounds, over the site of effusion (partially over the lower part of the back on the side of the effusion), instead of being diminished or absent become bronchial in character and harsher than normal. In such a case, the collapsed lower lobe of the lung is probably responsible for transmitting the sound from some large bronchus or bronchi to the fluid collection, even better than normally aerated lung tissue.

Medium-pitched bronchial breathing, clinically intermediate between cavernous and tubular breathing, when encountered over the chest suggests an incomplete loss of elasticity of the diseased lung as in some cases of tuberculous infiltration, bronchial carcinoma, fibrosis of lung and partial lung collapse.

Amphoric breathing is a special variety of high-pitched bronchial breathing with a distinctive "echo-like" or metallic quality that can be imitated artificially by blowing intensely across the mouth of a bottle or the open end of a rifle. It is usually indicative either of a large cavity in the lung with smooth walls, or of a pneumothorax communicating with a bronchus, in either case, the causative lesion, by amplifying or reinforcing certain vibrations or high-pitched overtones in the original bronchial sound, imparts to it an echo-like quality.

Bronchovesicular breathing A special variety of mixed breathing, combining some of the characteristics of both vesicular and bronchial breathing, is referred to as bronchovesicular or indeterminate breathing.

**CHARACTERISTICS** The characteristics of bronchovesicular breathing are ill-defined and variable in view of the mixed character of the breathing. As a general rule, the inspiratory phase is similar to that of normal vesicular breathing but slightly higher-pitched and of a muffled blowing quality. The expiratory phase is more intense, of a higher pitch, almost equal to inspiration and has a slightly tubular or hollow quality.

**SIGNIFICANCE** Normally, bronchovesicular breathing is heard over the upper part of the sternum in front because of the proximity of the large air passages, and in the interscapular region posteriorly because of bifurcation of the trachea.

Bronchovesicular breathing, when heard anywhere else over the chest, is usually indicative of disease. The causes of bronchovesicular breathing are more or less the same as those of bronchial breathing, being as a rule somewhat milder or less advanced. Patchy or incomplete consolidation of lung tissue, whether pneumonic, tuberculous or neoplastic in origin, frequently tends to bring about bronchovesicular breathing.

**MODE OF PRODUCTION** When consolidation of lung is incomplete (surrounded by areas of normal lung tissue), or patchy (interspersed with areas of normal lung tissue), the bronchial sounds which emanate from underlying bronchial tubes are modified during their transmission through normally-aerated parenchyma, becoming bronchovesicular in character

The bronchovesicular breathing heard in health over certain areas of the chest wall is due to close proximity of certain large air passages to the chest wall, small areas of normal lung tissue being interposed in between

**Metamorphosing breath sounds** Occasionally, the type of breathing may change suddenly in type, character or intensity, during one and the same breath. For instance, starting as a faint vesicular murmur during inspiration, the respiratory sound may change abruptly into a harsh and bronchial or amphoric type of breath sound. A metamorphosing breath sound is usually due to dislodgement of a mucus plug that is partially occluding a bronchus, by the incoming rush of air during inspiration.

### ADVENTITIOUS (ADDED) SOUNDS

Adventitious or added sounds are usually produced within the lung tissue, air passages or pleura. These true adventitious sounds must be clearly distinguished from so-called *extraneous* (*external, spurious, or accidental*) sounds, which are usually due to faulty auscultatory technique. The latter may be caused by (1) local contractions of chest muscles, secondary to chilling or nervous tension (muscle sounds), (2) friction of the chestpiece against the patient's skin or hair, (3) inadequate apposition of chestpiece against the chest wall, especially in thin-chested individuals with prominent ribs, or (4) extraneous noises from outside.

There are three principal sounds added to the breath sounds, (1) Wheezes or rhonchi, (2) Crackles or crepitations or rales, and (3) friction sounds or pleural rub.

**Wheezes** Wheezes or rhonchi or dry sounds are continuous musical sounds originating from the respiratory tract. Their significance is the same whether they are heard at a distance or only through the chest wall, the distinction between wheezing and rhonchi is superfluous.

**Mode of production** Wheezes are generated by air buzzing past airway distortions and secretions on the airway wall. The lung, it is suggested, can act like a reed of a wind instrument (a toy trumpet), sounding when the passage of air vibrates the wall (reed). A wheeze is produced as air is forced past a point at which opposing airway walls are just touching, these vibrate, generating the wheeze. This can occur in inspiration and expiration, more commonly the latter.

The vibratory reed analogy is perhaps the explanation for absence of wheeze in some patients with severe airways obstruction. The velocity of gas necessary to vibrate a reed is fairly high in presence of reduced ventilation or severe hyperinflation. Exhaled air will not reach a sufficient velocity to cause the obstructed airways to vibrate.

Pitch The pitch of the wheeze depends on — (a) the velocity of air flow at the generation point, and (b) the quality of the airway involved and hence the vibration frequency of the airway, (c) the airway calibre. The widely held belief that high-pitched sibilant rhonchi arise in small airways, while low-pitched sonorous rhonchi originate in large airways has been disproved. X

TYPES OF WHEEZES (a) Polyphonic wheeze The most common variety of wheezing is an expiratory musical sound containing several notes of different pitch audible at a distance. Healthy subjects can produce such a wheeze by a sudden violent expiratory effort. Polyphonic wheezing results from the oscillation of several large bronchi simultaneously brought to the point of closure by congestion of the mucous lining, contraction of smooth muscle and thickening of the layer of mucus. Because of expiratory narrowing at the airways there can be a large number of wheezes in expiration than inspiration. (b) Monophasic wheeze This is a single musical sound heard on inspiration or expiration in chronic bronchitis or emphysema. Each of these monophasic wheezes arises from a single airway brought to the point of closure by transmural pressure differences, swelling of the mucosa or bronchial constriction. Monophasic wheezes may be single or multiple, though their number is never very large. The wide conduction of these sounds — they are heard with varying intensity wherever the stethoscope is applied to the chest wall — produces the illusion of innumerable wheezes. Sometimes a single wheeze may be heard on inspiration. This can indicate structural change in an airway on the point of closure. Rarely it can indicate stenosis or foreign body or lymph-gland nearly occluding a principal bronchus, or may be the first sign of a bronchial carcinoma.

Monophasic wheezes are seldom loud enough to be heard with the unaided ear. An exception is stridor, a very loud monophasic musical sound produced by laryngeal or tracheal obstruction.

Crackles (rales, crepitations) Crackles are interrupted short sharp non-musical sounds.

Mode of production In the past, rales or crepitations have been said to arise from air bubbling through liquid in the airways and hence have been called 'moist sounds'. The reasons against this traditional explanation are that crackles may be — (1) present in inspiration only, sounds caused by air bubbling through fluid should be heard during both phases of respiration, (2) not shifted by coughing, (3) occur constantly in conditions such as pulmonary fibrosis in which there is no increase in secretions, (4) Recording of crackles (phonopneumogram) shows that individual crackles recur constantly in the same phase of ventilation during successive ventilatory cycles, as the same pressure and volume conditions are repeated, (5) Crackles are heard at the dependent lung bases in heart failure because the weight of the lung engorged by blood and tissue liquid partially deflates their lower parts. Lung inflation snaps open airways and crackling is heard. If crackles are auscul-

tated at the dependent right base, the patient standing, and the patient then leans forward, so that the right base is no longer dependent, there is no longer a column of liquid over the lung near the stethoscope, there is no partial deflation and no crackles can be heard at that point. Crackles return if the right base is once more made dependent by the patient standing upright again.

Crackles are miniature explosions that result from sudden equalisation of pressure when a closed airway separating two adjacent compartments of the lung, which contain gas under widely different pressures, suddenly opens. This occurs when a slightly occluded airway opens intermittently and allows bubbles of gas to pass, or in deflated territories of the lung where the airways remain closed until a late stage of inspiration. Crackles are characteristic of pneumonia, pulmonary fibrosis, bronchiectasis and pulmonary congestion. Common to all these is abnormal lung deflation, in which a number of airways are closed, or on the point of closure, at the start of inspiration. As inflation proceeds, the lung pulls one after another of these airways open, the liquid film sealing the airway is divided, the airway walls spring apart and air rushes in. These events are associated with a crackle, and a shower of crackles may be heard, as one after another of the previously closed airways is opened up as lung inflation proceeds.

#### VARIETIES OF CRACKLES

*Early inspiratory crackles* (Fig 10.31) Crackling heard early in inspiration are characteristic of widespread airway obstruction particularly chronic bronchitis. These crackles arise probably from the opening up of larger airways (in sequence according to their compliance or distensibility) closed by the air-trapping mechanism during the previous expiration. They are scanty, low-pitched, audible at the mouth as well as through the chest wall, and not posture dependant.

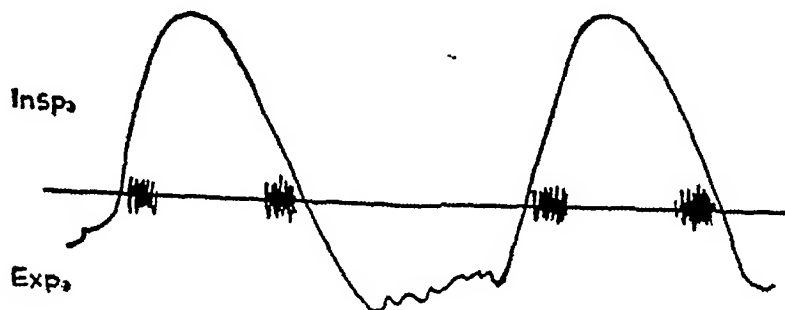


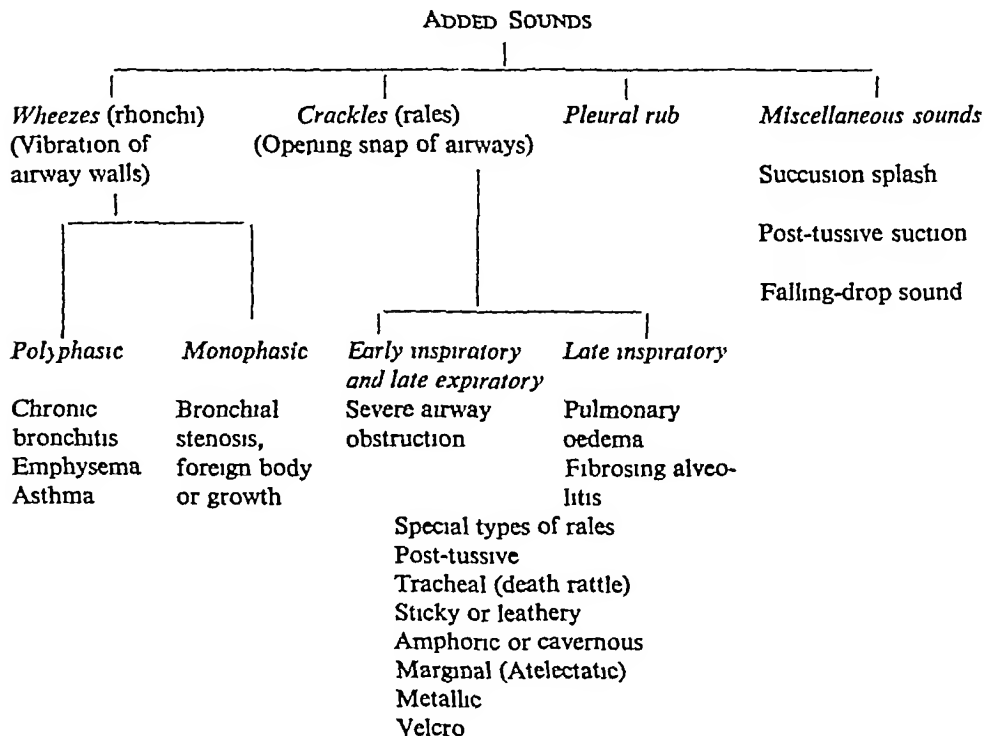
Fig. 10.31 Diagrammatic representation of early and late inspiratory crackles

*Late inspiratory crackles* (Fig 10 31) Crackles heard towards the end of inspiration are produced by delayed opening of the airways, and are heard at the base of both lungs in restrictive defects such as pulmonary oedema and pulmonary fibrosis, especially that due to fibrosing alveolitis and asbestosis. They are profuse, high-pitched and inaudible at the mouth and are modified by changes of posture such as bending forward or lying down, which often reduces their number or silences them altogether.

*Mid-inspiratory crackles* are probably characteristic of bronchiectasis.

*Expiratory crackles* These are characteristic of severe airways obstruction. They arise probably by the re-opening of airways, temporarily closed by the trapping mechanism as air is redistributed distal to larger and more proximal airways narrowed by the trapping mechanism during expiration. The expiratory crackling during this paradoxical flow in the expiratory cycle is repetitive but independent of posture. Copious secretions in the larger airways can give rise to clicking and bubbling noises. These are shifted by coughing.

**Crackle-wheeze.** A crackle immediately turning into a wheeze is sometimes heard towards the end of inspiration, especially in asthma. Inspiration just opens the airway with a crackle, but the opposing walls remain close enough to vibrate as air rushes in and a wheeze is heard. This supports the theory that the airways snap open to make a crackle and vibrate to wheeze.





**CONVENTIONAL CLASSIFICATION OF RALES** Since the newer above mentioned classification of rales may not suffice to explain all the facts, the traditional grouping of rales is worth mentioning

**Coarse rales** (Bubbling rales) Usually originating within large bronchial tubes, coarse rales occur during inspiration or expiration (most commonly at the end of inspiration), may be loud enough to be heard without a stethoscope or felt with the bare hand, and are either continuous or intermittent in occurrence. Coarse rales may be encountered in a variety of pathological conditions, including consolidation of lung, lung cavitation, lung abscess, pulmonary congestion or oedema and bronchiectasis. Coarse rales are frequently associated with rhonchi, as in case of bronchitis.

**Fine rales** (Crackling rales, *Crepitations*) Crepitant rales lack the bubbling character of coarse rales, displaying more of a "crackling" quality. This may be artificially imitated by rolling between the thumb and forefinger a lock of hair close to the ear or by separating moistened or sticky thumb and index finger near the ear, or by heating salt over a frying pan. Crepitations are usually very fine and multiple sounds which tend to occur transiently or evanescently at the end of inspiration either singly, in showers or fused into a single composite sound at the very end of inspiration. Crepitations are usually localized, constant and frequently accentuated by the act of coughing. They are due to the sudden separation of sticky alveolar walls, at the end of inspiration by the intruding of air, and are therefore indicative of fluid exudation, usually of inflammatory origin, within the alveoli.

Crepitations are particularly characteristic of the first stage of pneumonia (induced crepitations), early pulmonary tuberculosis, collapse or atelectasis of lung, bronchitis or pulmonary oedema (because of gravity, capillary filtration is greatest in lower zones, hence also the rales). Crepitant noises may be due at times to friction of the chestpiece of the stethoscope with a hairy chest wall, a source of diagnostic error that can be eliminated by shaving the hair off or by polishing it down with oil or water.

**Medium rales** Clinically intermediate in character and loudness between fine and coarse rales, medium-sized rales usually originate within small-sized bronchi or bronchioles and occur in bronchitis, fibrosis and at times bronchiectasis.

**MISCELLANEOUS FORMS OF RALES** A large number of special types of adventitious sounds of respiratory origin have been included under the generic designation of rales. Although some of these are distinctive enough to merit special consideration, the others are not characteristic enough to be described as special types. The commonly used terms "bubbling rales", "musical rales" or (rhonchi) and "crackling rales" (or crepitations) are synonymous with "coarse rales", "dry rales" and "fine rales" respectively.

**Post tussive rales** The presence of rales (or at times even the absence of rales) having been noted, the patient should be asked to cough once or twice at the end of inspiration and then immediately take a deep breath. The chest should again be carefully auscultated, particular

attention being paid to the phase of breathing from the end of the cough to the end of the following inspiration. Most latent or post-tussive rales are elicited during this interval.

*Tracheal rales* ("Death rattle") The largest and coarsest of the bubbling rales, tracheal rales, are usually heard over the trachea or lungs in seriously ill, comatose or markedly debilitated patients who are unable to cough out their respiratory secretions, the latter therefore accumulating within the trachea and air passages. By no means always pre-terminal, as at times believed, the tracheal rales may be loud enough to be heard at some distance from the patient without recourse to a stethoscope.

*Sticky or leathery rales* These are typical of bronchiectatic dilatations or cavities containing sticky exudate. Usually heard over one or both bases of the lungs, the characteristic nature of the rales may disclose the nature and site of the disease.

*Consonating rales* Highly resonant, moist and with a characteristic vibrant quality, this variety of rales is particularly characteristic of massive consolidation, as in lobar pneumonia, but may also be heard over the lung cavities, particularly of tuberculous origin.

*Metallic rales* (Tinkling consonances) These are highpitched, metallic and moist rales, which may be encountered in case of lung cavity or pneumothorax. The sounds are similar to those produced by drops of water falling from a height into a metal vessel.

*Velcro rales* Dry and leathery rales with cellophane-like quality heard most commonly in pulmonary interstitial fibrosis. The sound resembles that produced by tearing apart of matted strips of velcro adhesive.

*Subcrepitant rales* Intermediate in character and intensity between crepitations and medium-sized rales and somewhat difficult to distinguish from the former, are the so-called subcrepitant rales. These are usually produced within the smallest sized bronchi or bronchioles, and are indicative of fluid secretions in these air passages. They are particularly characteristic of lobar pneumonia (during phases of congestion and resolution), passive congestion of the lungs as in cardiac failure, tuberculous infiltration and acute pulmonary oedema (early phase).

Subcrepitant rales differ from crepitations in being lower in pitch, more intense, and moist instead of dry.

*Atelectatic rales* (Marginal rales) In elderly, debilitated or bed-ridden subjects, at the onset of forced or deep breathing, fine rales may be heard during inspiration over the bases and lateral margins of the lungs. Unlike crepitations, they tend to disappear after a few breaths and are usually associated with healthy lungs. They have been variously attributed to (1) traces of secretion within the alveoli and minute air passages (disappearing after the first few breaths), (2) uneven or delayed expansion of air-sacs within marginal and atelectatic basal areas of the lungs, (3) separation ("peeling off") of the diaphragmatic and costal layers of the pleura.

*Amphoric rales* (Cavernous rales) Peculiarly low or medium-pitched moist sounds with characteristic "echo-like" quality, these sounds suggest the presence of exudate-containing cavities within the lungs, communicating with bronchi.

*Indeterminate rales* When moist sounds fail to conform to the classical descriptions of the various types of rales, described above, they may be conveniently referred to as indeterminate or non-specific rales.

**Pleural rub (Pleural friction)** The commonest variety of adventitious sound originating in the pleural sac is the *pleural rub*

**MODE OF PRODUCTION** Normally, the parietal pleura slides smoothly and noiselessly over the visceral pleura due to the presence of a thin layer of lubricating secretion between the two layers

In the event of inflammation of the pleura, as in acute dry pleurisy, the two inflamed and roughened surfaces of the pleura rub against each other, giving rise to the characteristic friction rub

According to one hypothesis a pleural rub is generated as a bow vibrates a string. Acoustically it resembles a series of crackles of equal intensity and may be considered under the heading of a crackle

**SITE** The commonest site of pleural friction is the lower part of the axilla, the movement of the parietal over the visceral pleura being maximal in this area. At times, the friction sound is heard best elsewhere over the chest. It may be apical in pulmonary tuberculosis or even diffuse, involving the whole of one side of the chest

**CHARACTERISTICS** Pleural friction is usually easily diagnosable on the basis of the following features. The friction sound is characteristically (1) "rubbing" or "creaking" in quality, variously described as "grating", "leathery" or the "sound of new leather", a sound that can be artificially reproduced by placing the palm of one hand over the ear, the back of the hand being then rubbed with the fingers of the other hand, (2) interrupted or jerky in nature, (3) frequently loud in intensity, although the sound may at times be faint or only moderately loud, (4) superficial in character, sounding quite close to the ear, as if produced within the ear piece of the stethoscope, (5) accentuated, in many cases, by increased pressure of the chestpiece on the chest wall (causing further approximation of the roughened surfaces), (6) usually audible during both phases of respiration, although maximal during the phases of inspiration and expiration, when the pleural surfaces are actually rubbing against each other, (7) usually unaltered after bouts of coughing (unlike rales, which are often markedly influenced), (8) usually confined to a small or localized area of the chest (although rarely diffuse or generalized), (9) frequently palpable when coarse (*friction fremitus* or palpable pleural rub), at times the rub is audible and palpable even to the patient, (10) frequently associated with pain and local tenderness, (11) attenuated or eliminated transitorily at times, by the act of forced breathing, to reappear again during normal or shallow breathing

**DIAGNOSIS** Although, as a rule, clearly distinguishable from all other auditory phenomena of the chest, pleural friction may, at times, be confused with rales or crepitations, muscle sounds or pleuropericardial friction sounds

The following features are of value in distinguishing friction sounds from crackles or rales

<i>Pleural friction rub</i>	<i>Crackles or rales</i>
Usually superficial and loud	Not so superficial or loud
Continuous sound	Interrupted sound
Heard over a localised area	Heard over a wide area
Remains unaffected by coughing	Intensified or abolished by coughing
Pressure of chest-piece of stethoscope intensifies the sound	Pressure of chest-piece produces no effect
Associated with pain or local tenderness	No pain or local tenderness

A pleural friction sound can be easily distinguished from a pleuropericardial sound by the timing of the sound, the former being associated with the phases of respiration and the latter with the beating of the heart. Besides displaying a much slower rate or frequency than pleuropericardial friction, pleural friction is greatly influenced by the manner of breathing, being frequently eliminated by withholding the breath.

#### MISCELLANEOUS SOUNDS AND SIGNS

**Succession splash** (Hippocratic succession) This is a splashing sound which can be heard over the chest either with the stethoscope or with the unaided ear applied to the chest wall or at some distance from it (Fig 10 34) when the chest of the patient is shaken suddenly by the examiner with his hands. The sound may be loud enough to be appreciated by the patient himself, and may even be felt by the examiner's hands. Normally heard over the abdomen (stomach or colon), filled with fluid and gas, a succession splash over the chest is always pathological, being indicative of (1) fluid and air in the pleural cavity as in hydro- or pyo-pneumothorax, (2) herniation of stomach or colon into the thoracic cavity through the diaphragm, or (3) a large cavity containing fluid and air in the lung.

**Forced expiratory time (FET)** This gives a rough estimate of the severity of airways obstruction. The patient is asked to expire forcibly with the mouth open after a full inspiration while the examiner listens over the trachea with a stethoscope. Normally this takes less than 4 seconds. A value of 6 seconds or more indicates airway obstruction.

**Clicking sounds**. A small left sided pneumothorax may at times give rise to clicking sounds in tune with the heart beat. These are best heard usually to the left of the sternum, during expiration and with the patient turning to the left. The sound is due to systolic contraction of the heart alternately closing and opening a small, overlying artificial pleural pocket. Occasionally a sound resembling a clicking sound (particularly after coughing) may be due

to the heart beat producing a wave in a small hydropneumothorax. Rarely a clicking sound may result from slipping of a displaced costal cartilage at the eighth or ninth rib. The click may be induced by breathing or with movement.

**Mediastinal crunch (Hamman's sign)** In mediastinal emphysema, especially, with left sided pneumothorax, systolic crunching sounds may be heard coinciding with systole over the mediastinum along the left sternal edge from third to fifth interspaces with the patient in sitting position. It may result from sudden movement of air by the systolic contraction of the heart or sudden contact and separation of the two pleural layers. Such loud crepitant sounds are however not diagnostic of mediastinal emphysema and may be heard in pneumothorax in absence of mediastinal emphysema, bullous emphysema of lingula, lower oesophageal dilatation, dilatation of stomach or pneumoperitoneum with ascent of the left diaphragm.

**Post-tussive suction** (Post-tussive splash or succussion) A characteristic medium or low-pitched "sucking" sound, audible over the chest wall, during the long inspiration following a bout of coughing, is referred to as post-tussive suction or splash. It is indicative of a thin walled and compressible lung cavity in communication with a bronchus. During coughing, the collapsible thin-walled cavity is deprived of its air content, re-entry of air into the empty cavity through a bronchus during the post-tussive act of inspiration brings about the characteristic suction sound.

**Falling drop sound** (Metallic tinkling) A highly resonant and reverberating metallic sound or tinkle, heard over the chest, and resembling the tolling of a distant church-bell or the sound of drops of water falling into a partially filled metallic cistern. The metallic tinkle can be induced by changing the posture from recumbent to sitting-up or by laughing, coughing or forced breathing. Whenever present, the sign is suggestive of (1) hydropneumothorax or (2) large cavity in the lung, containing fluid and air. It may be heard normally over the abdomen, in case of gastric or intestinal distension.

Metallic tinkling may be due to one of two possible mechanisms: (1) drops of fluid falling from the lung border on to a fluid level (falling drop sound), or (2) bursting of bubbles on the surface of the fluid (bursting bubbles sound).

**Water whistle sound** In the event of a pulmonary fistula or fistulous opening below the level of fluid, in case of hydropneumothorax, resulting at times accidentally through a needle-puncture, a peculiar bubbling or splashing sound with a metallic quality, the so-called "water-whistle" sound, may be heard.

## VOICE SOUNDS (VOCAL RESONANCE)

By vocal resonance (or voice sounds) is meant the sounds heard over various parts of the chest during the act of speech or phonation. Vibrations initiated by the spoken voice or whispered voice are transmitted along the air passages and through the lung parenchyma to the chest-wall, resulting in certain auditory or auscultatory sound phenomena, referred to as either "spoken voice sounds" or "whispered voice sounds". Vocal resonance is the auscultatory equivalent of the palpatory phenomenon of tactile vocal fremitus, the same laws govern the mode of production, transmission, elicitation and abnormalities associated with the two phenomena.

**Spoken voice sounds** **MODE OF PRODUCTION** In the case of spoken voice, the fundamental tones of the "voice sound" are initiated within the larynx by vibrations of the vocal cords, certain tones or overtones of the "laryngeal sound" are then intensified or modified by the oral cavity, acting as a resonating chamber in conjunction with the nasal and laryngeal cavities, depending on alterations in shape and size of these resonating cavities, tones of certain wave-length are selectively accentuated, thus modifying the original character of the glottic sound. Similar accentuations or modifications are also effected but to a lesser degree by the thorax and air passages acting as resonators. The process of selective accentuation or modification of the glottic sound is referred to as articulation.

**TRANSMISSION** The vibration of the vocal cords, which accompanies initiation of the glottic sound, sets the entire bronchial tree and broncho-pulmonary column of air into vibration. Voice sounds are usually better heard over the trachea than bronchi and over larger bronchi than over the smaller ones. This is because "loudness" and degree of transmission of such a sound through an air passage are directly dependent on (1) proximity to larynx, (2) proximity to chest wall, (3) calibre, and (4) cartilaginous structure.

**TECHNIQUE** For eliciting spoken voice sounds, auscultation of symmetrical areas of the two sides of the patient's bared chest is carried out with the aid of a stethoscope, whilst the patient is made to repeat over and over again, in a slow, loud, uniform and deep voice, some convenient stock phrase, such as "ninety-nine" or "one-one-one" or "one-two-three". Normally, spoken voice sounds are heard over the chest as weak, muffled, and indistinct rumbling sounds, with individual syllables blurred and indistinguishable, this is referred to as normal vocal resonance. Over the trachea and larynx, the voice sounds are normally much louder and clearer (laryngophony or physiological bronchophony).

**PHYSIOLOGICAL VARIATIONS** Vocal resonance is a somewhat variable phenomenon, even in health, being greatly dependent on the age, sex and state of nutrition of the patient, on the pitch of the patient's voice, and on the region or site of chest auscultated, and being proportional to the resiliency of the chest wall and elasticity of lung tissue. Vocal resonance, as a rule loudest in childhood and weakest in old age, because of the deeper and lower-pitched nature of the male voice, is usually louder in men than women. Thin-chested or under-nourished individuals usually yield better voice sounds than the thick chested, stockily built or obese.

**REGIONAL VARIATIONS** Vocal resonance varies widely over the different regions of the chest, even in health. Thus, it is normally louder on the right side of the chest than on the left, louder anteriorly than posteriorly, very loud over the suprasternal region and interscapular regions (owing to proximity of the air passages), weak over the mammary, scapular and infrascapular

regions, and absent over the inferior sternal region. Vocal resonance is loud over the seventh cervical vertebra and can be heard as far down as the fifth dorsal vertebra in health.

**SPOKEN VOICE SOUNDS IN DISEASE** Vocal resonance may be pathologically altered in one of several ways during states of disease. Thus, it may be (1) increased or loud, (2) diminished or weak, (3) absent, (4) loud and clear (bronchophony), (5) clear and articulate or syllabic in character (spoken pectoriloquy or "chest speech"), (6) nasal or bleating in character (aegophony), (7) ringing or metallic and echoing in character (amphoric voice sound or resonance).

*Increased vocal resonance* This is noted over areas of consolidation or infiltration of lung tissue, solidified lung being a good conductor of sound vibrations. It may also be noted over a small superficial lung cavity, or over an area of partial atelectasis, bronchiectasis or compensatory emphysema. The presence of tumour, lymph gland or adhesive band between a bronchus and the chest wall may increase vocal resonance locally by transmitting the vibrations better to the chest wall. Occasionally, spoken voice sounds are paradoxically accentuated over a pleural effusion, when the subjacent lung tissue becomes solidified, with the bronchus supplying the part remaining patent.

*Decreased or diminished vocal resonance* Usually associated with defective production or defective transmission (or both) of sound vibrations, decreased vocal resonance may be encountered in case of partial obstruction of an air passage (as in partial laryngeal stenosis or glottic oedema), hypertrophic emphysema, thickened pleura, small pleural effusion, partial pneumothorax, oedema of chest wall, or malignant disease of pleura. A feeble voice, from disease or debility, may also result in decreased vocal resonance.

*Absence of vocal resonance* Due to absence of production or transmission of sound vibrations, loss of voice sounds may be noted in case of deaf-mutism, vocal cord paralysis, large pleural effusion, massive emphysema, pneumothorax, acute pulmonary oedema or absence of lung tissue (as in diaphragmatic hernia or after lobectomy).

**BRONCHOPHONY** When spoken voice sounds appear unduly loud or intense, clear and sound close to the ear ("chest voice"), the individual words or syllables however remaining indistinguishable, the condition is referred to as bronchophony. Normally audible over the larynx and trachea (laryngophony or physiological bronchophony), true bronchophony is never heard over the chest or lung parenchyma, except in case of disease. Pathological bronchophony is usually indicative of (1) lung consolidation as in lobar pneumonia (red or grey hepatization), massive bronchopneumonia, caseous or tuberculous pneumonia, (2) compressed lung tissue, as in pleural effusion.

(above the level of fluid) or intrathoracic tumour (causing lung compression), (3) tuberculous or bronchiectatic lung cavity, usually located superficially or surrounded by consolidated lung tissue

**SPOKEN PECTORILOQUY** When spoken voice sounds appear unduly loud, clear and syllabic, individual words or syllables being clearly distinguishable, the condition is referred to as spoken pectoriloquy. There is a clear transmission of syllabic or articulate speech ("chest speech") directly into the ear. Pectoriloquy may be secondary to spoken or whispered speech, the latter variety being usually of greater diagnostic value. The causes of spoken and whispered pectoriloquy being identical, they are considered together under the caption of whispered pectoriloquy.

**AEGOPHONY** When spoken voice sounds during auscultation display a peculiar quivering, nasal quality, like the "bleating of a goat" or "speech with the nostrils compressed", the condition is referred to as aegophony.

Aegophony may be due to (1) pleural effusion, being usually heard best along the border of a pleural effusion, just above the upper level of percussional dullness, (2) over a pleural effusion overlying an area of lung consolidation, or (3) over a cavity half-filled with secretion. It is usually heard best over the back, close to or lateral to the lower angle of the scapula. The mode of production of aegophony remains obscure, it has been attributed either to (1) the interposition of a thin layer of fluid between the lung and chest wall, allowing the transmission of overtones but damping off the lower fundamental tones or to (2) a partial compression of lung tissue underneath the upper part of the effusion, altering the normal relationship between bronchi and lung parenchyma and thus causing a reinforcement of high-pitched nasal sounds.

Although stressed by some as diagnostic of pleural effusion and of value in differentiating an effusion from a lung consolidation, aegophony need not necessarily be present over a pleural effusion, and may occasionally be encountered over a lung consolidation. Hence its presence is neither necessary for, nor diagnostic of, pleural effusion.

**Amphoric voice sounds** (Echoing resonance) When spoken voice sounds, besides being intense and clear, display a characteristic metallic, *echoing* character, they are referred to as amphoric voice sounds. They can be artificially imitated by speaking into the open mouth of a jar.

They are characteristically observed in case of either (1) a large cavity with tense or rigid walls communicating with a bronchus or (2) over an open or valvular pneumothorax.

**The scratch sign** This is of help in the diagnosis of pneumothorax. It is elicited by placing the stethoscope at some midline position on the chest, either over the spine or sternum. At equidistant points from the stethoscope the skin is scratched with a finger or blunt object, and the sound from the two areas compared. A positive sign consists of a considerably louder and harsher sound on the side of the pneumothorax. The difference in sound is attributed to high-frequency sound absorption by the subadjacent normal lung of overtones produced by the scratch.



**Whispered voice sounds** Sounds heard over the chest wall during the act of "whispering" (*whispered voice*) on the part of the patient are referred to as whispered voice sounds.

**MODE OF PRODUCTION.** Unlike spoken speech, the glottis or vocal cords play no part in the production of the fundamental tones of whispered voice sounds, the larynx being passive. The phenomenon of articulation takes over the entire function of sound production. Also, in the case of whispered voice sounds, there is no vibratory tremor or sympathetic vibration on the part of the thoracic walls, as in the case of spoken voice sounds.

**METHOD OR TECHNIQUE.** The method of eliciting whispered voice sounds is to make the patient repeat over and over again, in a whisper, some convenient stock phrase, such as "one, one, one", "one, two, three", or "ninety-nine", during which time auscultation of various parts of the chest wall is carried out in a systematic and symmetrical manner.

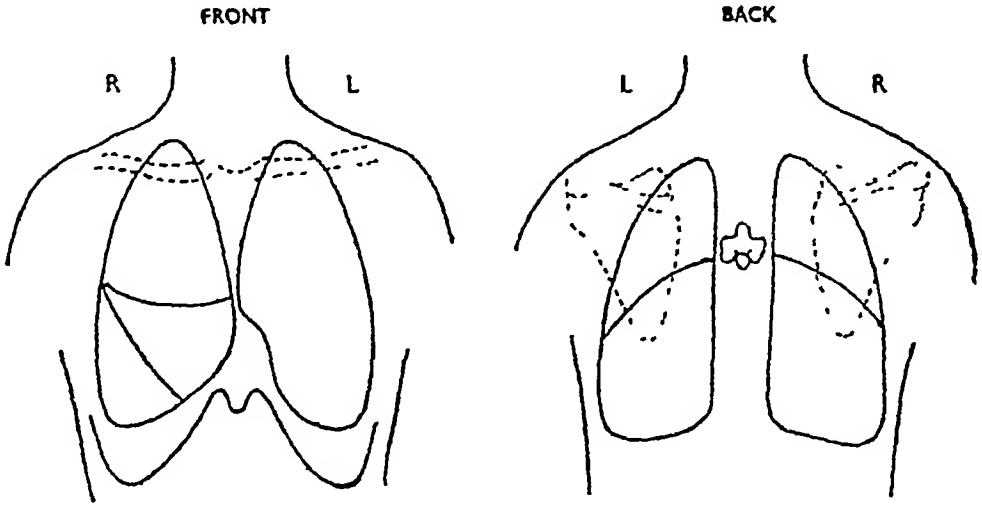
**WHISPERED VOICE SOUNDS IN HEALTH** Normally, whispered voice sounds are heard very indistinctly or faintly over certain areas of the chest only, viz over the main air passages (such as the trachea and bronchi), over the spine of the seventh cervical vertebra, over the inner end of the second right intercostal space and in the interscapular region. The whispered voice is usually inaudible over normal lung parenchyma, especially at the bases. The presence of whispered voice sounds, therefore, anywhere over normal lung tissue is suggestive of disease.

**Whispered voice sounds in disease** When the whispered voice becomes audible over areas of the chest wall (e.g. over normal lung tissue), where it is normally inaudible or becomes more intense than normal, it is usually indicative of patchy consolidation, infiltration or distension of lung tissue. Thus, it may be encountered over areas of bronchopneumonia, tuberculous infiltration or compensatory emphysema.

**WHISPERED PECTORILOQUY** (Whispering pectoriloquy, bronchial whisper) When the whispered voice is transmitted to the chest wall with sufficient clarity, to maintain its articulate or syllabic character, the individual syllables or words being clearly distinguishable as if uttered directly into the examiner's ear, the condition is referred to as whispered pectoriloquy. A sign of great diagnostic value, whispering pectoriloquy is indicative of (1) a fairly large cavity in the lung, communicating with a bronchus, (2) a massive or diffuse consolidation of lung tissue overlying or adjacent to a bronchus, (3) retracted or partially compressed lung tissue just above (or at times below) the level of a pleural effusion, or (4) obstruction of a large bronchus by a tumour (the sign being positive just proximal to the site of obstruction).

According to some, the presence of whispered voice sounds over lung tissue is often an earlier, more sensitive and dependable sign of early pneumonia,

NAME \_\_\_\_\_ DATE \_\_\_\_\_ RECORD NO \_\_\_\_\_



### INSPECTION

Respiratory movements zone of normal, diminished or absence of excursion of chest to be shown both in front and back

Apex beat position to be indicated by dark spot

### PALPATION:

Vocal fremitus

Increased VF +

Diminished VF —

Absent VF Abs

### PERCUSSION

Impaired note |||||

Dull note |||||

Flat note |||||

### AUSCULTATION:

Breath sounds

Normal

Diminished

Absent Abs Br

Exaggerated

Bronchial

Cavernous

Broncho-vesicular

Adventitious sounds

Rhonchi

Coarse rales

Medium rales

Fine rales

Pleural friction

Sig \_\_\_\_\_

Fig 10 32 Graphic recording of physical signs in the respiratory system and the symbols which may be used

consolidation or infiltration of the lung than bronchial breathing, a view not shared by all. However bronchial breathing and whispering pectoriloquy are inseparable physical signs.

Whispered voice sounds are of particular value in serious cases of lobar pneumonia and lung cavitation, where, because of grave illness, exhaustion or pain, normal breath sounds are not clearly audible, and forced respiration and elicitation of spoken voice sounds are not advisable.

**D'ESPINE'S SIGN.** Normally, whispered voice sounds are audible over the spines of the lower cervical vertebrae, not being transmitted, however, to below the level of the seventh cervical vertebra in infancy and childhood, and below the level of the third thoracic vertebra in adults. When whispering pectoriloquy is encountered below these levels, d'Espine's sign is said to be "positive." A positive d'Espine's sign is frequently, but not invariably, suggestive of (1) enlarged mediastinal or tracheobronchial lymph nodes, (2) tumour in the posterior mediastinum, or (3) a patch of central pneumonia.

**BACELLI'S SIGN** (Amphonic pectoriloquy) This sign was used once to distinguish purulent from serous pleural effusions. Transmission of whispered voice sounds through pleural effusion was supposed to rule out a purulent and favour a serous effusion in the pleural cavity. This sign has become more or less obsolete.

**ARTIFICIAL VOCAL RESONANCE (Phlegmaphonia)** This may be resorted to in rare cases of deaf-mutism, aphonia or coma, where spoken or whispered voice sounds are either unobtainable or inadvisable. Sengerwald's technique consists in a repeated percussion or tapping of the patient's thyroid cartilage (the mouth of the patient being kept closed), the examiner listening at the same time to various places on the patient's chest.

**Graphic recording of auscultatory findings** As in the case of cardiovascular auscultatory findings, different methods of recording respiratory signs graphically have been recommended. Fig. 10 32 illustrates one such method. Such graphic representation permits a rapid visualization of the respiratory findings at a glance.

# 11 | The Abdomen

It is not always possible, in the examination of the abdomen, to adhere strictly to the usual order of eliciting physical signs, viz inspection, palpation, percussion, and auscultation, as in the case of the cardiovascular and respiratory systems. It is however advisable, even in the case of the abdomen, to resort to some systematic plan of examination, in order to avoid important omissions.

**Topographical anatomy** The surface markings or abdominal projections of intra-abdominal organs are not as precise or fixed as those of intrathoracic organs, they being dependent to a great extent on the constitution, state of nutrition and musculature of the individual.

## IMPORTANT ABDOMINAL LANDMARKS

Certain artificial and skeletal landmarks are of invaluable service in localizing symptoms, signs, structures or organs, within the abdominal cavity, in relation to the anterior abdominal wall.

By the projection of certain vertical and horizontal lines, the abdomen can be arbitrarily divided into quadrants or regions. Such a division serves to facilitate the task of locating the exact site of a pain or an intra-abdominal structure or organ in relation to the abdominal wall.

The simplest method is to divide the abdomen into four quadrants, viz the right and left upper and the right and left lower quadrants, by drawing two lines, vertically and horizontally, through the umbilicus. This method of division does not permit a delineation of certain important regions of the abdomen, such as the epigastric and the suprapubic regions.

A better plan is to subdivide the abdomen into nine regions or segments, with the aid of two vertical and two horizontal lines or landmarks. The vertical line on either side is drawn upwards through the mid-inguinal point, or midway between the midline of the abdomen and the anterior superior iliac spine. The upper horizontal line crosses the abdomen along the lowest levels of the costal margins or tenth costal cartilages, whilst the lower horizontal line connects the two anterior superior iliac spines. By this method, it is possible to map out nine important regions or segments of the abdomen (Fig 11.1).

Certain important intra-abdominal contents or viscera, in relation to these arbitrary divisions of the abdomen, must be borne in mind

1 <i>Right hypochondriac</i>	2. <i>Epigastric</i>	3 <i>Left hypochondriac</i>
Right lobe of liver	Pyloric end of stomach	Stomach
Gall-bladder	Duodenum	Spleen
Part of duodenum	Pancreas	Tail of pancreas
Hepatic flexure of colon	Aorta	Splenic flexure of colon
Part of right kidney	Part of the liver	Upper part of the left kidney
Right suprarenal gland		Part of the left lobe of the liver (at times)
4 <i>Right lumbar</i>	5 <i>Umbilical</i>	6 <i>Left lumbar</i>
Ascending colon	Omentum	Descending colon
Lower half of right kidney	Transverse colon	Lower half of left kidney
Part of duodenum and jejunum	Lower part of jejunum and ileum	Portions of jejunum and ileum
7 <i>Right iliac</i>	8 <i>Hypogastric</i>	9 <i>Left iliac</i>
Caecum	Ileum	Sigmoid colon
Appendix	Bladder	Left ureter
Lower end of ileum	Gravid pregnant uterus	Left spermatic cord in male
Right ureter		Left ovary in female
Right spermatic cord in male		
Right ovary in female		

NATURAL LANDMARKS, which can also be usefully employed for locating symptoms or signs in relation to the abdominal wall, are the xiphisternum or ensiform cartilage, the umbilicus or navel, the symphysis pubis, right and left costal margins, right and left anterior superior iliac spines, the linea alba and the linea semilunaris

The linea alba or white line is a vertical tendinous line extending from the tip of the xiphoid or ensiform cartilage to the symphysis pubis. The linea semilunaris is a curved line along the outer border of the rectus muscle on either side, extending from the ninth costal cartilage to a point midway between the umbilicus and the anterior superior spine of the ilium

## INSPECTION

### METHODS

For a proper inspection of the abdomen, the lighting must be adequate and tangential, daylight being preferable to artificial lighting. Inspection may be carried out with the patient either recumbent or standing, the former being the position of choice for most cases

Inspection of the *recumbent patient* is carried out with the patient lying comfortably on his back, with one pillow under the head. The examiner either sits or stoops by the side of the bed in order to bring his eyes on a level with

the anterior abdominal wall. Alternatively, he may stand by the side or at the foot of the bed and inspect the patient's abdomen, tangentially, from a higher level, viewing the abdomen in this manner may serve to bring out minor variations of abdominal contour and reveal the presence of abdominal lumps.

An inspection of the abdomen with the patient *erect* or *upright* is also advisable in most cases, this position frequently facilitates the detection of visible herniations, engorged veins, visceroptosis or of a pendulous abdomen.

An examination of the *back*, below the level of the tenth rib, should be routinely included in abdominal inspection, abnormalities of contour in this region may suggest the presence of intra-abdominal disease.

#### SCHEME OF INSPECTION

The following must be routinely looked for during inspection of the abdomen: (1) the contour or shape, (2) abdominal asymmetry through swelling or retraction, (3) the state of the skin, (4) the umbilicus, (5) the outlines of the recti muscles, (6) the veins, (7) the arteries, (8) peristaltic movements, and (9) respiratory movements.

**Contour or shape.** There are three main types of abdominal contour. In the *flat* type of abdomen, which is common in young adults, the rib margins and anterior abdominal wall are more or less on the same level. In the *globular* or *round type*, the anterior abdominal wall presents a forward convexity, usually through obesity or lack of muscle tone. In the *scaphoid* or "boat-shaped" abdomen, which is common in thin subjects and those suffering from wasting diseases, cachexia, dehydration and meningitis, the anterior abdominal wall presents a forward concavity or sunken appearance.

A symmetrical distension or bulging of the abdomen may be due to obesity, gaseous distension, ascites or pregnancy.

**Abdominal asymmetry.** Lack of symmetry of the abdomen may be due to local bulging or retraction of the abdominal wall, the former being far more common.

*A localized swelling*, bulging or protrusion of the abdominal wall may be due to enlargement of a solid organ such as the liver (Fig. 11.2) or spleen, in which case the lower border of the organ usually moves up and down with respiration. Whilst in the case of liver enlargement the swelling is on the right side or middle of the abdomen, in the case of splenic enlargement it is on the left side, and in the case of an enlarged kidney it is located either anteriorly, posteriorly or in the flank. In a thin subject, even an enlarged gall-bladder may show itself as a visible protuberance in front.

Undue distension of the stomach, small intestine or colon may be obvious on inspection, the prominence being in the epigastric and left hypochondriac regions in the case of the stomach (Fig. 11.3), over the central or umbilical

region (Fig 11 4) in the case of the small intestine, and along one or other side, below the costal margin, in case of the colon

A tumour or cyst arising from the pancreas, retroperitoneal tissue or mesentery may cause abdominal asymmetry through local bulging. A greatly distended urinary bladder, from retention of urine, may cause a visible swelling in the suprapubic region. A fullness or prominence of the right or left costo-vertebral angle of the back may be caused by a tumour, by infection within or outside the kidney or a cold abscess (Fig 11 5).

*Local retraction* of the abdominal wall may be due to injury to some underlying muscle or to the displacement of an organ, such as the liver. Moderate retraction of both hypochondriac regions is common in visceroptosis. In acute intestinal obstruction, the area beyond the seat of the obstruction frequently appears retracted.

*State of the skin.* Visible linear scars over the abdominal wall are usually indicative of previous abdominal operations. The abdomen may be a mass of surgical scars from previous laparotomies in Munchausen's syndrome. Branding marks of diverse sizes and shapes and "stellar" leech-marks are at times observed in this country, they usually suggest the existence of some abdominal pain or distress in the past. A dry, tense, glistening and transparent abdominal skin, through stretching, suggests chronic distension of the abdomen. White lines over the lower part of the abdomen, the so-called linear albicantes with an associated loss or rupture of elastic fibres, are evidence of previous stretching of the skin, through pregnancy, ascites, abdominal tumour, cyst or massive enlargement of an organ such as the spleen, rapid weight gain and in Cushing's syndrome. Prolonged over-stretching of the abdominal skin, as in pregnancy, may result in a deposition of pigment along the *linea alba*, the so-called *linea nigra*. Purplish striae over the abdomen are suggestive of adrenocortical hyperfunction of Cushing's syndrome. Tinted striae have been reported to occur in young obese individuals of both sexes with increased urinary steroid excretion. Multiple small nodules of the skin (*carcinoma cutis*) in the region of the umbilicus, caused by metastatic deposits, may be the first sign of intra-abdominal malignancy.

*State of the umbilicus.* Normally indrawn or flush with the skin surface, the umbilicus may alter in shape or position, depending on the tone of the abdominal muscles and the degree of abdominal distension. In ascites, it may become transversely stretched (smiling umbilicus), everted or ballooned out. In ovarian cyst it tends to be vertically stretched.

A bluish discolouration of the umbilical area (Cullen's sign) secondary to blood-pigment reaching the umbilicus through the lymphatics of the medial umbilical ligament, is suggestive of intra-abdominal haemorrhage, usually from ruptured ectopic pregnancy, it is a sign usually of ominous significance.

A cherry-red swelling of the umbilicus is usually inflammatory and suggestive of an inflamed Meckel's diverticulum.

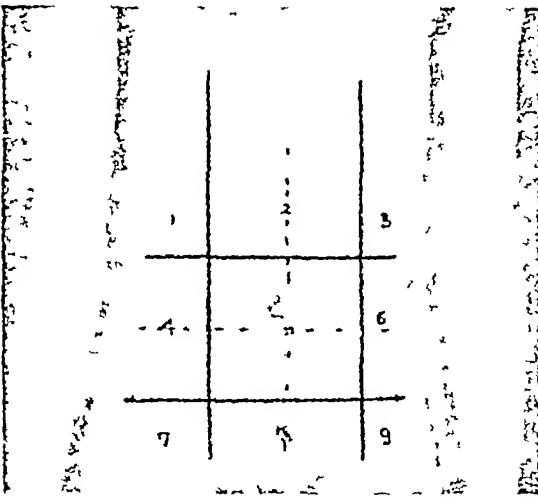


Fig 11.1 The regions of the abdomen



Fig 11.2 Liver abscess producing right upper abdominal bulge

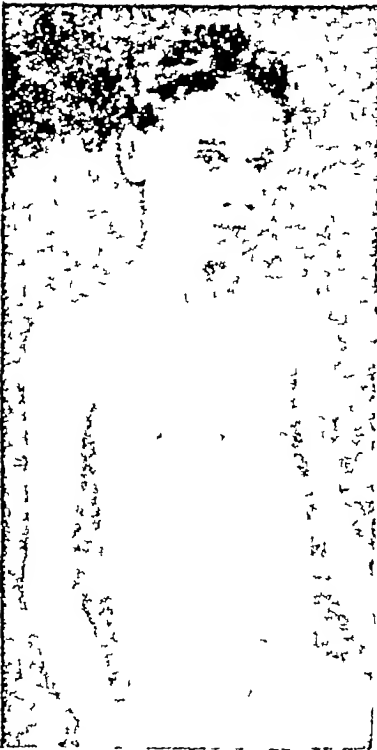


Fig 11.3 Asymmetrical distension of abdomen from pyloric obstruction



Fig 11.4 Ventral hernia, local bulging of abdomen



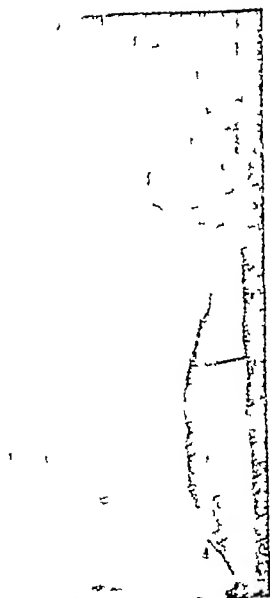


Fig 11.5 Cold abscess in right loin

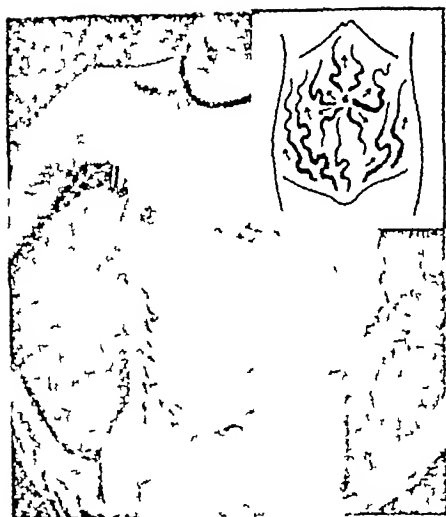


Fig 11.6 Prominent anterior abdominal veins due to both portal and inferior vena caval obstruction Insert In portal obstruction the veins radiate from the umbilicus in obstruction of inferior vena cava the collateral venous channels carry the blood upwards



Fig 11.9 Visible peristalsis in pyloric obstruction Ether may be sprayed on the abdominal wall to incite peristalsis



Fig. 11.10 Ladder pattern resulting from intestinal obstruction

Fig 11.7, see p 373  
Fig 11.8, see p 374

An umbilical discharge, serous or seropurulent, may be due to bacterial or fungal infection of the umbilicus or a patent urachus. Faecal fistulae at the umbilicus may be due to patent omphalomesenteric duct, inflammatory changes commencing in the intestine and extending up to the umbilicus, carcinoma of an abdominal organ (usually of the stomach reaching to and breaking through the umbilicus), or inflammatory conditions of the umbilicus extending to and involving the intestine.

**Diastasis of the recti.** Marked weakness of the recti muscles may lead to their wide separation in the midline, either above or below the level of the umbilicus. Such a diastasis or divarication of the recti may not be apparent unless the patient raises his head and shoulders in the lying-down position, or stands up and strains. Ascites is another cause of divarication. In severe cases, part of the intestine may protrude forwards, through the gap between the recti muscles.

**Veins.** Prominence or distension of the superficial veins of the abdominal wall is abnormal and indicative of obstruction to the return circulation through the portal vein or the inferior vena cava (Fig 11 6), this may result from an enlarged liver, intra-abdominal tumour, chronic distension of the stomach or mediastinal new growth. The normal flow of blood in the superficial veins of the abdomen is from the umbilicus upwards to the thorax, and from the umbilicus down to the groin (Fig 11 7).

In *intra-hepatic portal venous obstruction* or portal hypertension, as a rule secondary to cirrhosis, only a few veins are usually visible in the epigastric region, although large and tortuous veins radiating from the umbilicus (*caput medusae*) may be observed in some cases, the blood flow in these veins is directed radially away from the umbilicus (Fig 11 6 *inset*). In *extra-hepatic obstruction*, the flow is towards the umbilicus (Fig 11 7) because the para-umbilical vein conveys blood from the abdominal wall to the liver.

*Inferior vena caval obstruction* also gives rise to collateral veins (Fig 11 7) which can be differentiated from portal collateral veins clinically thus

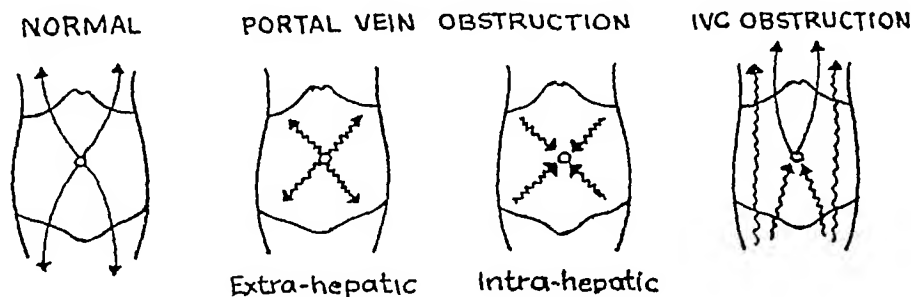


Fig 11 7 Diagram of venous return from lower chest and abdomen, normal and abnormal

*Portal collaterals*

- 1 Prominent collateral veins radiating from umbilicus
- 2 No dilated vessels over the back
- 3 Direction of flow downwards in vessels below umbilicus
- 4 Bruits common over dilated vessels

*Vena cava collaterals*

- 1 Collateral tend to be more prominent in the flanks
- 2 Often dilated vessels over back
- 3 Direction of flow upwards in vessels below umbilicus
- 4 Bruits not heard over dilated vessels

To determine the direction of blood flow in a collateral vein, a part of the vein which is free from branches for a distance of 3 to 4 cm is selected. The examiner's two fingers are pressed close together over the mid-section of this vein and then drawn apart in order to empty a section of the vein of blood, the pressure with the fingers being maintained (Fig 11.8), one finger is then lifted at a time and the direction of filling up of the emptied section of vein is observed. However

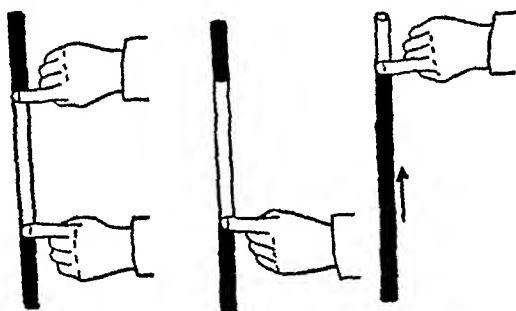


Fig 11.8 Method of determining direction of blood flow in collateral veins

the valves in dilated veins may become ineffective and it may not be always possible to ascertain the direction of blood flow in a vessel by the technique of stripping it between two fingers. Whilst in health the flow of blood over the lower two-thirds of the abdominal wall is directed downwards, in inferior vena caval obstruction the direction of flow is upwards or in a reverse direction, part of the blood from the lower part of the body finds its way, through the collateral circulation of the abdominal parietes, to the superior vena cava.

The degree of distension of the subcutaneous veins over the abdomen is not necessarily proportionate to the height of intra-abdominal pressure or to the degree of obstruction to venous return, because of the possibility of anastomotic veins elsewhere being more dilated than those of the subcutaneous tissues.

In massive ascites, both the portal and the inferior venal collaterals may be visibly dilated, when the intra-abdominal tension is reduced in such a case, the inferior vena caval collaterals usually disappear but the portal collaterals tend to persist.

**Arteries** A normal abdominal aorta may pulsate visibly in case of a scaphoid abdomen, aortic regurgitation, neurocirculatory asthenia, thyrotoxicosis and lordosis of the upper lumbar region, the aorta overriding the spine

being thrust forwards. A pulsatile swelling in the epigastrium may be due to an aneurysm of the abdominal aorta, but is more often due to an epigastric tumour or a mass of enlarged lymphnodes overlying or attached to the aorta, when such a tumour is adherent to the aorta, the pulsations persist even with the patient in the knee-chest position, with a non-adherent lump, on the other hand, the pulsations tend to disappear. Abdominal pulsations may also be caused by an enlarged and pulsating liver.

**Peristalsis.** Abdominal peristalsis may be visible in perfectly normal individuals with thin abdominal walls and in cachectic or malnourished children. Apart from these exceptional instances, visible peristalsis is always indicative of bowel obstruction. To look for peristalsis the abdomen must be carefully observed for several minutes, a gentle flicking of the abdominal wall or the pouring of a drop or two of ether over it may serve to initiate visible peristaltic waves.

The site, direction and nature of the peristalsis are helpful in determining the site of obstruction. In pyloric obstruction (Fig 11 9) the peristalsis is best marked in the umbilical area, starts as a depression in the left hypochondrium from behind the left costal margin and moves towards the right. In small intestinal obstruction, the peristalsis is most obvious below the umbilicus, in the form of worm-like, undulatory movements proceeding in various directions. In obstruction close to the caecum, the peristalsis is best seen in the umbilical region, the distended coils of intestine appear as parallel ridges one above the other, forming a so-called "ladder pattern" (Fig 11 10), a sign almost diagnostic of advanced intestinal obstruction. When the transverse colon is obstructed, peristaltic waves, travelling from right to left, are seen either in the epigastric region or below the umbilicus, depending on the position occupied by this part of the bowel. In obstructive lesions of the ascending or descending colon, the peristaltic wave travels vertically upward or downward in the flank and mimics an alternately appearing and disappearing mass or tumour. In colonic obstruction it is common to find the caecum visibly and palpably distended and rising with each wave of peristalsis.

**Respiratory movements.** Normally, the abdominal wall moves freely with respiration in males (abdominal breathing) but not in females (costal or thoracic breathing). Restriction of the respiratory movement of the abdominal wall in a male may be due to (1) inflammation of the peritoneum, or (2) referred pain from an inflammatory lesion of the diaphragmatic pleura or an abdominal muscle, (3) a tense ascites, or (4) a massive intra-abdominal cyst or tumour. A large tumour or cyst may offer a mechanical impediment to movement. A diminution of respiratory movement of the abdominal wall in a restricted area, such as the right upper or lower quadrant, suggests inflammation of an underlying viscus, such as the gall-bladder or the appendix.

## PALPATION

Palpation is by far the most informative method of examining the abdomen. It may yield information of great diagnostic value.

**Technique.** Abdominal palpation is possible with the patient either standing, sitting-up or recumbent, the ideal position being the dorsal recumbent posture, with the patient lying flat on the back and with one pillow under the head. A flexion of the patient's legs, with the soles of the feet resting on the bed, is advisable, in order to secure adequate abdominal relaxation. The patient is then asked to breathe quietly through the nose, whilst the arms lie loosely by his sides. The examiner either sits by the side of the bed on a chair or stool or bends down sufficiently when standing to bring himself on a level with the patient. The palpating hand must be warm in order to prevent a reflex contraction of the abdominal muscles, if not, palpation may be carried out with the patient's shirt, underwear or a bed-sheet interposed between the hand and the abdominal wall. The examiner's forearm and hand must be on a level with or slightly below the anterior abdominal wall. Palpation, whether carried out with the palm of the hand or the fingers, must be gentle but firm, avoiding sudden thrusts. Although the various regions of the abdomen can be palpated in any order, it is best to proceed systematically, leaving palpation of tender or painful areas, which are likely to yield involuntary protective spasms of the abdominal wall, to the very last.

In case of inadequate relaxation of the abdominal wall, as in the case of nervous patients, the patient's attention may be diverted elsewhere by engaging him in conversation during palpation, at the same time, his face is observed closely for any wincing movement, which might suggest the presence of a tender area. Another method of overcoming muscular rigidity, in refractory cases, is to exert pressure, with the base of the palm of the left hand, on the lower part of the patient's sternum, thus immobilizing the thorax and forcing the patient to breathe abdominally, the examiner then seizes the opportunity of palpating the abdomen, with his right hand, during each phase of inspiration, when the abdominal muscles are relaxed.

Palpation of the abdomen in the *knee-chest* or *knee-elbow* position is reserved for special cases only, for palpating small lumps not clearly demonstrable in the dorsal decubitus, for demonstrating small intra-abdominal fluid collections, and for differentiating pulsatile tumours from abdominal aneurysms, such a posture may prove invaluable.

## METHODS OF PALPATION

Four main methods of abdominal palpation are in use, viz. superficial palpation, deep palpation, bimanual palpation, and the method of dipping or ballotment.

(1) **Superficial palpation** A gentle or light superficial palpation of the abdomen is advisable, to begin with, in all cases, it serves to allay the patient's fear of being hurt, secures adequate muscle relaxation, and enables the examiner to obtain a general idea about the abdomen. Except for a few hypersensitive individuals, who display ticklishness or resistance to palpation, the abdominal wall in most subjects is soft and yields readily to superficial palpation. Elicitation of any degree of tenderness or muscle-guarding, during light palpation, is indicative of inflammation, enlargement or new growth affecting an underlying viscus. The order in which the various areas of the abdomen are palpated depends on circumstances, as a rule, however, it is best to leave the area under suspicion to the last. Thus, with a history suggestive of cholecystitis, palpation of the right upper quadrant or gall-bladder area should be carried out last. A circumscribed area of tenderness may be associated with a hypersensitiveness or hyperalgesia of the overlying skin, this can be demonstrated by pinching the skin and subcutaneous tissues with the fingers.

(2) **Deep palpation** This may be carried out by the examiner, using either *one* hand or *both* hands. During routine deep palpation, the right hand of the examiner is placed flat over the patient's abdomen, and palpation carried out with the volar surfaces of the flexed fingers, a steady and firm pressure being maintained throughout, sudden or jerky movements with the finger-tips must be avoided at all times.

In the case of a massive, tense or thick-walled abdomen and for palpating a deeply-placed structure within the abdominal cavity, *re-inforced* palpation, using both hands, is preferable. In this method, the fingers of one hand are placed loosely over the region to be palpated and pressure exerted upon them with the distal ends of the fingers of the other hand. The patient is then asked to breathe in and out deeply, and whilst the abdomen falls in during expiration, the palpating hands are made to sink deeper and deeper into the abdomen, until the desired organ or mass within the abdomen is felt by the palpating or passive hand.

(3) **Bimanual palpation** Particularly useful for the palpation of kidneys, this method may also be employed to advantage in the case of the liver and spleen. The examiner, with his face directed towards the patient, stands or sits on the patient's right side. For palpation of the kidney, he places one hand under the loin with the tips of the extended fingers resting against the erector spinae muscle, the other hand being placed flat over the abdomen with the fingers pointing upwards towards the costal margin. The patient is then asked to take a deep breath whilst firm pressure is maintained with both hands. When a kidney is enlarged or palpable, it moves downwards into the gap between the examiner's hands and separates them.

(4) **Dipping or ballotment** In the presence of a massive ascites or large amount of free fluid within the abdomen, ordinary palpation may fail to reveal the presence of an enlarged organ or pathological mass, in such a case, the

method of *dipping* may be resorted to. Either the tips of the examiner's extended fingers are suddenly pushed into the abdomen with a quick stabbing movement or, alternatively, the extended hand is suddenly flexed at the metacarpophalangeal joints in order to depress the abdominal wall, and then equally quickly withdrawn. Such a procedure, by displacing the fluid momentarily, permits the fingers, after a series of such movements, to reach and feel any solid organ or lump that may be masked due to the intra-abdominal fluid.

**NORMAL STRUCTURES** which may be felt during abdominal palpation are the following: (1) The abdominal aorta—the systolic thrust of the abdominal aorta, lying against the bony spine in a thin or hypersensitive individual, may be easily palpable. (2) The bodies of the lower lumbar vertebrae, particularly in cases of lordosis or curvature of the lumbar spine. (3) The lower border of the liver during deep inspiration, particularly in emphysematous subjects. (4) The right kidney—in thin individuals with lax abdominal walls, in multiparous women and in the aged, the lower pole of the kidney may be felt during palpation at the height of inspiration, the right kidney, being somewhat lower than the left, is usually felt in such cases. (5) The transverse colon—in youthful and elderly subjects with lax abdominal walls, the transverse colon may be palpable, as a transversely placed elongated structure, just above the umbilicus.

#### ABNORMAL PALPATORY FINDINGS

**Muscle guarding or rigidity.** Any inequality in the resistance offered by the abdominal musculature, on the two sides, during palpation, must be carefully noted. An increased resistance or *rigidity*, when not induced voluntarily, is abnormal.

A *generalized rigidity* over the entire abdomen is strongly suggestive of general peritonitis, in such a case, the rigidity is "board-like" and even firm pressure may fail to indent the wall even slightly. Diffuse rigidity may also be encountered in cases of acute intestinal obstruction, meningitis, tetanus, tabetic crisis, lead colic, spinal injury, marked abdominal distension or extensive intra-abdominal adhesions or psychopathic drug addiction. *Localized or unilateral rigidity* may be due to local inflammation as in appendicitis, or to the presence of a solid organ or tumour directly underneath the abdominal wall. *Transient or momentary rigidity* may be encountered, in sensitive individuals, during attacks of severe abdominal pain.

**Tenderness.** Pain on pressure, or tenderness during palpation is always suggestive of disease or inflammation of an underlying organ, provided local tenderness of the intervening abdominal wall can be ruled out. The phenomenon of *rebound tenderness* can be elicited by exerting firm pressure with the hand over a painful area and then releasing the pressure suddenly, in which case the patient may complain of sharp pain. Rebound tenderness signifies inflammation not only of the visceral but also the parietal peritoneum. Re-

bound tenderness may also be elicited at times in association with distension of bowel loop or loops. *Shifting tenderness* is a useful sign for differentiating abdominal tenderness due to acute nonspecific mesenteric adenitis from that due to appendicitis. In the former, after laying the patient on the left side for a few minutes, the maximum tenderness moves to the left of the original site.

When pressure applied to one area of the abdomen elicits pain or tenderness in another area, the phenomenon is spoken of as *referred* or *crossed* tenderness. Thus in a case of acute appendicitis, pain or tenderness in the right lower abdominal quadrant may be induced by pressure on the descending colon (Rovsing's sign). When a circumscribed area of tenderness is elicited by light or deep palpation, one must examine the overlying skin for hypersensitiveness.

**Oedema of abdominal wall.** This can be demonstrated either by digital pressure, which leads to the phenomenon of pitting or by picking up a fold of skin and subcutaneous tissues in which case the thickening of tissues through oedema becomes obvious. Oedema of the abdominal wall may be associated with generalized oedema or anasarca, as in cases of nephritis or cardiac failure, or with massive ascites. In myxoedema, although the abdominal wall feels thickened, there is no pitting on pressure.

**Doughy feel.** A doughy or resilient feel, when encountered during palpation of the abdomen, is said to be diagnostic of tuberculous peritonitis. It may afford useful confirmatory evidence of the disease. A doughy feel may be encountered at times in tropical sprue and in multiparous obese females.

**Haematoma of rectus sheath.** A tender mass in the hypogastrium to the right or left of the midline associated with pain, necessitates exclusion of a haematoma of the rectus sheath. Predisposing causes are pregnancy, trauma to the abdomen, severe coughing or use of anticoagulant drugs. A useful sign is that when the recti are contracted by sitting, the mass is still felt and fixed, while an intra-abdominal mass is no longer easily palpable (Fothergill's sign).

#### INVESTIGATION OF A MASS OR LUMP

A palpable intra-abdominal mass or lump may be caused by the enlargement of a solid organ, distension of a hollow viscus, presence of a new growth or tumour, or a mass of inflammatory tissue. The following observations are necessary in order to determine the nature of an abdominal lump.

(1) **Position or anatomical relationship.** To determine whether a lump is intra-abdominal or extra-abdominal, the recumbent patient is asked to raise his head and shoulders, if the lump becomes more prominent, it is within the abdominal wall, if it tends to disappear, it is intra-abdominal. As a rule, the presence of a lump in any part of the abdomen suggests disease of some underlying organ or structure in the same region, occasionally, however, a tumour or abscess originating in one part may grow or advance in such a manner that it becomes palpable elsewhere.



(2) **Size.** The size of a lump may at times suggest its true nature, for instance, whilst a huge liver is more in favour of malignancy than cirrhosis, a massive spleen suggests leukaemia or chronic malaria rather than Hodgkin's disease

(3) **Consistency** Whilst a malignant tumour is usually firm, hard or stony, a cyst, inflammatory swelling or tumour undergoing softening or necrosis tends to be resilient, faecal masses are usually indentable

(4) **Surface and contour** A smooth surface with a rounded contour may be caused by the enlargement of a solid or hollow organ. In diffuse enlargements of solid organs, the surface may be either smooth, granular or nodular. In carcinoma of the liver, the surface is frequently studded with large, umbilicated nodules

(5) **Tenderness** Unless the patient is unduly nervous or sensitive, abdominal tenderness usually suggests an inflammatory or congestive condition of some underlying structure. For instance, tenderness of the liver is more suggestive of hepatitis or the passive congestion of heart failure than of cirrhosis or malignant infiltration.

(6) **Movement with respiration** The respiratory movement of an intra-abdominal lump can be determined by resting the fingers against the lower border of the lump, whilst observing the excursions of these fingers during deep inspiration and expiration. A tumour that moves freely with respiration probably arises from an organ in close relationship to the diaphragm, such as the liver, spleen or, to a lesser extent, kidney. Immobility of the tumour, on the other hand, suggests its origin from a hollow organ, the presence of adhesions, fixation to retroperitoneal tissues, or diaphragmatic fixation through pleurisy, emphysema or massive enlargement of the liver or spleen

(7) **Pulsation** A visible or palpable abdominal pulsation, transmitted from the abdominal aorta through a solid mass or tumour is not uncommon, it is nonexpansile in character unlike an aneurysm of the abdominal aorta. A pulsatile swelling in the epigastrium should suggest an aneurysm of the abdominal aorta, or epigastric tumour, or enlarged lymph nodes overlying or attached to the aorta. If the pulsation is due to a tumour not adherent to the aorta, it will disappear if palpation is made with the patient in the knee-chest position

(8) **Mobility** The mobility of a lump can be determined by gently pushing it in various directions, and observing the range of movement in each direction, a floating kidney, for instance, can be most readily pushed upwards and backwards. A lump that can be freely moved about during palpation and tends to drop in the upright posture is suggestive of a ptotic or floating kidney, liver or spleen, a tumour of the pylorus or intestine, a faecal mass or gall-stones. A slightly mobile lump is likely to be associated with the gall-bladder, omentum, uterus or ovary. A fixed or immovable lump suggests tumour of the pancreas,

aneurysm of abdominal aorta, appendicular abscess, cold abscess or enlargement of retroperitoneal glands. The position of a tumour arising from the stomach or intestine may alter from time to time with peristalsis.

(9) **Fluctuation** A fluctuating mass is usually suggestive of a cyst containing fluid or semifluid, as in hydronephrosis, pyonephrosis, ovarian cyst, hydatid cyst, distended bladder, encysted abscess, hydramnios or ectopic gestation.

## PALPATION OF THE LIVER

**Regional anatomy** The liver is the largest glandular organ of the body. It occupies the right upper quadrant of the abdomen and extends for a variable distance into the left upper quadrant (Fig. 11.11). Except for the epigastric region, where the liver comes into direct contact with the anterior abdominal wall, it is covered by ribs or costal cartilages. The upper border of the right lobe corresponds to the level of the fifth rib, an inch medial to the midclavicular line, or a point about one centimetre below the right nipple. The upper border of the left lobe is at the level of the sixth rib in the left midclavicular line, or about two centimetres below the left nipple. The lower border of the liver extends obliquely upwards from the ninth right to the eighth left costal cartilage, crossing the middle about half way between the base of the xiphoid cartilage and the umbilicus. The left lobe extends to the left of the sternum for about five centimetres.

**Technique** Palpation of the liver should start laterally near the right anterior axillary line and away from the right rectus muscle in order to avoid its tendinous intersection being mistaken for the liver edge. To avoid overlooking gross liver enlargements, it is advisable to start palpation low down in the right iliac fossa and then gradually work upwards until an increased sense of resistance is encountered. During palpation, the patient is asked to take deep breaths, the liver edge moves downwards, from one to three centimetres, during deep inspiration in a normal subject, and even more in an emphysematous subject, the liver being pushed down by the low position of the diaphragm. In most cases of generalized enlargement of the liver, the left lobe is easily felt in the epigastrium, somewhere between the xiphoid and umbilicus. Sometimes, as in hepatic syphilis (*hepar lobatum*), there is a disproportionate enlargement of the left lobe of the liver. Riedel's lobe, a downward tongue-like projection of the right lobe of the liver, is a simple anatomical structure, which may at times be mistaken for a pathological lump.

## ENLARGED OR PALPABLE LIVER

**INVESTIGATION** In case of palpable or enlarged liver, the following features must be investigated systematically.

(1) *Size or degree of enlargement* with reference to the costal margin. This is expressed either as so many "finger-breadths" or "inches" below the right costal margin, or as a slight (+), moderate (++) or massive (+++) enlargement, a mild degree of liver enlargement is common in viral hepatitis, a moderate degree in hepatitis, cirrhosis and obstructive jaundice, and a massive or gross enlargement, reaching below the umbilicus, in secondary malignant

deposits, leukaemia, Hodgkin's disease, amyloidosis, congestive cardiac failure, and gross fatty infiltration as in alcoholics

(2) *Shape* of the palpable portion of the liver Whilst a rounded mass is suggestive of an abscess or hydatid cyst, a preponderant enlargement of the left lobe of the liver suggests hepatic syphilis, a primary or metastatic tumour or an abscess

(3) *Edge or border* The liver edge must be carefully palpated all along its length to determine if it is regular or irregular, firm or soft, rounded or sharp Whilst in congestive heart failure, the edge is firm and smooth, in constrictive pericarditis it is hard

(4) *Surface* The anterior surface of the liver must be carefully palpated, during inspiration, for smoothness, granularity or irregularity of surface, and for the presence of nodules or tumour projections The surface of the liver is usually smooth in amoebic or viral hepatitis, liver abscess, congestive heart failure and amyloid disease, granular in hepatic cirrhosis, and nodular and umbilicated in malignancy Massive nodules are suggestive of the liver of tertiary syphilis (*hepar lobatum*)

(5) *Tenderness* A tender liver is usual in viral hepatitis, amoebic hepatitis, liver abscess, congestive heart failure and infected cyst A malignant liver is as a rule non-tender, unless haemorrhage or necrosis has occurred, a cirrhotic liver is always non-tender Shock tenderness on first percussion over the right lower ribs posteriorly is an important sign of hepatic inflammation

(6) *Consistency* The liver usually feels soft in case of fatty infiltration or amyloid disease Whilst a soft liver, in association with portal hypertension, suggests extra-hepatic portal venous obstruction, a firm liver supports a diagnosis of cirrhosis In malignancy and cirrhosis, the liver is characteristically hard on palpation

(7) *Movement on respiration* A failure on the part of the liver to move with respiration suggests its fixity or adhesion to another structure or structures

(8) *Pulsation* Pulsation of the liver, like neck-vein pulsation, represents the pressure waves transmitted to it from the right atrium of the heart, in the case of the liver, however, they are transmitted through the inferior and not superior vena cava Pulsation of the liver is particularly common in tricuspid incompetence and usually occurs during ventricular systole, it is best felt by bimanual palpation, with one hand over the right lower ribs posteriorly and the other over the liver in the right upper quadrant, anteriorly, the two hands tend to separate with each expansile pulsation of the liver A second method of feeling liver pulsation is to maintain pressure with the closed fist over the ribs overlying the anterolateral surface of the liver, with each pulsation, the fist moves upwards and outwards Similarly the tips of two index fingers can be placed over the liver when the expansile nature of the pulsation can be noted

A transmitted pulsation can be distinguished from an intrinsic pulsation of the liver by timing it with the pulsation of the carotid artery in the neck, whilst transmitted pulsation is synchronous with carotid pulsation, intrinsic liver pulsation, as in tricuspid insufficiency, usually follows carotid pulsation. In case of subsequent development of cardiac cirrhosis, liver pulsations tend to diminish or disappear. A highly vascular tumour of the liver is another cause of liver pulsation.

(9) *Change in size* A rapidly enlarging mass of the liver is highly suggestive of malignancy or amoebic abscess. A rapid decrease in liver size may be noted in case of congestive heart failure, obstructive jaundice or severe diabetes, during the phase of treatment or recovery. A decrease in size of the liver despite persistent heart failure, or a fixed enlargement of the liver in spite of clearing up of heart failure suggests the development of cardiac cirrhosis.

(10) *Hepatic rub* A palpable rub over the right hypochondrium while the patient takes a deep breath suggests carcinoma of the liver and perihepatitis.

**Palpable liver. SIGNIFICANCE** A palpable liver does not necessarily mean an enlarged liver or hepatomegaly. Palpability of liver well below the costal margin may be due to ptosis, downward displacement or enlargement of the liver.

(1) *Ptosis of liver* In visceroptotic individuals, a ptosis of the liver or hepatoptosis is frequently associated with ptosis of other intra-abdominal viscera. Whilst the lower edge is palpable well below the costal margin, the upper border of the liver is also displaced downwards, as demonstrable by percussion or fluoroscopy. A ptosed liver, being mobile, can often be pushed back into normal position by upward pressure on the lower edge, it tends, however, to regain its ptosed position in the erect posture.

(2) *Downward displacement* of the liver may be due to right-sided pleural effusion, empyema, pneumothorax or massive pericardial effusion. Pulmonary emphysema may also cause it by its flattening effect on the diaphragm. The liver may occasionally be displaced downwards in case of severe kyphoscoliosis or diaphragmatic hernia.

### (3) *Enlargement of liver*

**CAUSES OF HEPATOMEGALY** A. *Infection* (i) Viral infective hepatitis, yellow fever (ii) Bacterial tuberculosis, typhoid (iii) Spirochaetal syphilis, leptospirosis (iv) Protozoal amoebiasis, malaria, kala-azar (v) Parasitic hydatid disease (vi) Mycotic actinomycosis, histoplasmosis

B. *Circulatory disturbances* (i) Local congestion portal obstruction (ii) General congestion congestive cardiac failure

C. *Toxic hepatitis* Anaesthetic agents, cinchophen, sulphonamides, dilantin, arsenic, bismuth and gold

D. *Reticulo-endothelial diseases* (i) Primary reticulosis leukaemias, Hodgkin's disease, lymphosarcoma, multiple myeloma (ii) Lipoid storage disease xanthomatosis, Gaucher's

disease, Niemann-Pick disease (iii) Infectious diseases infectious mononucleosis, hepatic granuloma

E. *Biliary obstruction* biliary calculi, tumour or stricture of the bile duct

F. *Metabolic disorders* diabetes mellitus, glycogen storage disease, haemochromatosis

G. *Tumours and cysts* benign or malignant hepatoma, benign or malignant cholangioma, secondaries, polycystic disease, non parasitic cyst, hydatid cyst

**DIFFERENTIAL DIAGNOSIS** The following conditions may have to be clinically differentiated from hepatic enlargement

(1) *Malignancy of stomach* A massive infiltration of the stomach, particularly of the greater curvature, by malignant disease may simulate enlargement of the liver. The abdominal mass, although usually palpable in the epigastrium, may also be felt in the left upper, right upper or even a lower quadrant of the abdomen, depending on the location of the tumour within the stomach and of the stomach within the abdominal cavity. A stomach tumour is usually movable, tender and tends to display visible peristalsis, even when its borders are ill-defined.

(2) *Carcinoma of colon* A tumour arising from the transverse colon may at times simulate liver enlargement. It is, however, characteristically hard, nodular and mobile (unless fixed by adhesions) whenever palpable, in case it causes intestinal obstruction, there is usually audible or visible peristalsis with abdominal distension or tympanitis.

(3) *Kidney tumour* A massive tumour arising from the right kidney is usually smooth and rounded, gives a tense or elastic feel to the palpating hand, occupies a lateral position, and is bimanually palpable and ballotable.

(4) *Omental mass* A thickened or rolled up greater omentum, secondary to tuberculous peritonitis, usually presents a tender, sausage-shaped lump, which hardly moves with respiration, there is usually a band of resonance demonstrable between it and the lower border of the liver, whilst tuberculous glands are frequently demonstrable elsewhere within the abdomen.

## PALPATION OF SPLEEN

**Regional anatomy** The spleen lies behind the 9th, 10th, and 11th ribs, with its long axis along the line of the 10th rib. Anteriorly it extends to the midaxillary line, while posteriorly its superior angle is one and a half inches lateral to the 10th thoracic spine. It is separated from the 9th, 10th and 11th ribs by the diaphragm, the lower part of the left lung and the costodiaphragmatic recess of the pleura. The spleen follows closely the respiratory excursions of the diaphragm. It is normally not palpable, in order to become palpable, the spleen must enlarge to about twice its normal size.

**Technique** Several methods are used for palpating the spleen. (1) The usual or classical method is to palpate the spleen with only one hand. With the patient lying on his back with drawn-up knees, the examiner stands or sits on the patient's right side, facing him. With the flat of the right hand palpation begins in the right iliac fossa, moving gradually upwards and obliquely until the sharp margin of the enlarged organ is felt. (2) Palpation of the spleen may also be carried out in the dorsal recumbent position, bimanually, with the left hand of the examiner resting posteriorly over the left lumbar region, and the right hand placed flat over the left hypochondrium with the fingers pointing towards the spleen. The patient is asked to take a deep breath, and whilst with the left hand gentle pressure is exerted to push the intra-

abdominal structures anteriorly and medially, the right hand is made to dip in a little, the spleen, when enlarged, comes and hits the finger tips (3) A third method of palpation, particularly useful for minor degrees of splenic enlargement, is to stand, facing the foot of the bed, on the left side of the patient. The hooked fingers of the examiner's left hand are placed under the costal margin, whilst with the right hand pressure is exerted against the posterolateral aspect of the lower thorax, in order to displace the spleen forward. The patient is then asked to take a deep breath, and the spleen is felt at the end of inspiration (4) In yet another method, help is taken of gravity to bring the spleen forwards. The patient lies on his right side with the knee flexed and a pillow underneath the body, between the right iliac fossa and the costal margin to obtain full relaxation. The hooked fingers of the examiner's right hand are placed under the left costal margin and the patient asked to breathe in and out deeply.

(4) *Manoeuvre of Bockus* In patients with *splanchnoptotic habitus* in whom ptosis or enlargement of spleen is suspected but palpation by ordinary methods has failed to feel the spleen, the patient (if not ill) is made to jump up and down a few times and then made to lie down in the recumbent or right lateral position. The spleen edge may now be palpable.

**Palpable spleen INVESTIGATION** When a spleen is palpable, the following features about the enlargement must be noted

(1) *Situation* Depending on size, an enlarged spleen may occupy, besides the left hypochondrium, the left lumbar and umbilical regions of the abdomen.

(2) *Size* Enlargement of spleen is described either in terms of "finger-breadths" or "inches" below the costal margin, or as mild or just palpable (+), moderate (++), or massive (+++), when the spleen extends to the umbilicus or beyond. The degree of enlargement of the spleen may even suggest the nature of the disease. Whilst a mild degree of enlargement is common in acute malaria, typhoid fever and subacute bacterial endocarditis, a massive spleen suggests leukaemia, chronic malaria or kala-azar.

(3) *Shape* The normal pyriform or triangular shape of the spleen is usually maintained in disease, in the case of new growth, however, it may be grossly distorted.

(4) *Consistency* The spleen is usually *soft* in typhoid fever, septicaemia and subacute bacterial endocarditis, *firm* in cirrhosis and myeloid leukaemia, and *hard* in chronic malaria.

(5) *Surface* In most cases, the spleen has a smooth or even surface.

(6) *Edge* One or more notches are usually palpable along the medial or anterior margin of the spleen, a feature of some diagnostic value.

(7) *Depth* A splenic tumour being very superficial, it is usually impossible to invaginate one's fingers between the left costal margin and the spleen.

(8) *Tenderness* With the exception of abscess formation or infarction of spleen, a splenic mass is usually non-tender.

(9) *Movement with respiration* The spleen moves freely with respiration except when adherent or massive, it tends to move downwards and to the right during inspiration, unlike the liver and kidney which move directly downwards

(10) *Rub* A splenic rub, due to perisplenitis, when felt or heard over the spleen is suggestive of splenic infarction

(11) *Relation to spinal muscle* Because of a gap or space between the posterior border of the spleen and the erector spinae muscle, it is possible to push one's fingers in behind a splenic tumour

CAUSES OF SPLENOMEGALY The main causes of splenic enlargement are

1 *Infections* (a) Acute—Typhoid fever, septicaemia, acute bacterial endocarditis, viral hepatitis, infectious mononucleosis (b) Subacute and chronic—Malaria, kala-azar, subacute bacterial endocarditis, tuberculosis, brucellosis, bilharziasis

2 *Circulatory*—(a) Congestive cardiac failure (b) Portal hypertension (i) Suprahepatic Blocked hepatic veins (Budd Chiari syndrome) High pressure in inferior vena cava—Constrictive pericarditis, tricuspid valve disease. (ii) Intrahepatic Obstruction to Portal and hepatic veins in liver—Cirrhosis, congenital hepatic fibrosis, schistosomiasis, and sarcoid (iii) Extrahepatic—Obstruction to portal vein outside liver (iv) Increased blood flow—A-V aneurysm of splenic vessels

3 *Inflammatory and collagen disorders* Systemic lupus erythematosus, Felty's syndrome

4 *Haematological* (a) Acute leukaemias, chronic myeloid leukaemia, chronic lymphatic leukaemia, lymphomas (b) Myeloproliferative disorders—polycythaemia vera, primary myeloid metaplasia. (c) Haemolytic disorders—hereditary spherocytosis, thalassaemia.

5 *Storage diseases*—Gaucher's disease, Niemann-Pick disease, histiocytosis X

6 *Tumors and cysts*

7 *Miscellaneous*—Iron deficiency anaemia, pernicious anaemia, amyloidosis, myeloma, mastocytosis

DIFFERENTIAL DIAGNOSIS OF SPLENIC ENLARGEMENT The following conditions may offer diagnostic difficulties

(1) *Enlarged or displaced left kidney* This may be distinguished from an enlarged spleen by attention to the following points

<i>Spleen</i>	<i>Kidney</i>
1 Sharp edge	1 Rounded edge
2 A characteristic notch	2 No notch
3 Angular poles	3 Rounded poles
4 Superficial	4 Deep
5 Fingers cannot be pushed between spleen and costal margin	5 Possible to push fingers between kidney and costal margin
6 Dull on percussion	6 Vertical band of colonic resonance in front
7 Bimanually not palpable	7 Bimanually palpable
8 Moves freely with respiration	8 Movement with respiration not so marked
9 Direction of movement on respiration downwards and to the right	9 Moves directly downward on respiration

- |   |   |
|---|---|
| 10 Tendency to bulge forwards and inwards<br>11 The loin (posteriorly) is resonant on percussion<br>12 Cannot be pushed back into the loin<br>13 Fingers can be dipped into space between lump and erector spinae muscles posteriorly | 10 Tendency to bulge into the loin<br>11 No area of resonance between the mass and the spine, the whole loin being dull<br>12. Can be pushed backwards into the loin<br>13 No space between lump and erector spinae muscles posteriorly |
|---|---|

(2) *Liver enlargement* A greatly enlarged left lobe of the liver may simulate a splenic swelling. The enlargement in such a case is usually tender, a notch is absent, the surface may be irregular, and the lump is continuous with the liver.

(3) *Omental mass* A rolled-up carcinomatous or tuberculous omentum may be mistakenly diagnosed as an enlarged spleen. The edge of such a lump is not well defined, a notch is usually absent (although a notch may be simulated when there is fusion of more than one lump), there is a gap between the lump and costal margin, and a band of resonance usually exists between the mass and the normal area of splenic dullness, similar lumps may also be palpable in other parts of the abdomen.

(4) *Carcinoma of stomach* The edge of a carcinomatous lump involving the body of the stomach is not well defined, a notch is absent. It extends more to the right than upwards and a band of resonance is usually demonstrable in front of the lump.

(5) *Suprarenal tumour* The physical signs in the case of a malignant suprarenal mass may be closely similar to those of a kidney tumour, being more deeply placed, it is easier to invaginate one's fingers between a suprarenal lump and the costal margin.

(6) *Carcinoma of colon* A carcinomatous mass involving the splenic flexure is ill-defined, a notch cannot be felt, the position of the lump tends to vary from day to day, and intestinal symptoms are usually conspicuous.

(7) *Pancreatic tumour* Commonly situated in the midline, a pancreatic cyst may be large enough to occupy a major portion of the abdominal cavity. A pancreatic lump or tumour, unlike a splenic mass, displays no notch, usually crosses over to the right of the midline above the umbilicus, is usually more nodular and is likely to be associated with jaundice.

(8) *Ovarian tumour* The spleen may be large enough to reach down to the pelvis and simulate a tumour of the ovary. An ovarian tumour rarely attains the level of the ribs, a gap being palpable between its upper border and the costal margin, it does not move with respiration and can be felt to rise from the pelvis. The lower part of the abdomen appears more prominent than the upper, the tumour is more globular in shape, has an ill-defined edge and no notch can be felt. The tumour extends more to the right of the midline than an enlarged spleen and is more likely to transmit aortic pulsations. A vaginal examination confirms its ovarian origin.

(9) *Faecal accumulations* The splenic flexure or adjacent part of the transverse or descending colon when loaded with faeces has been mistaken for an enlarged spleen. Such a mass is however more irregular and cylindrical in shape and can be indented by the finger.

#### PALPATION OF GALL-BLADDER

**Regional anatomy** The gall-bladder is a pear-shaped, saccular structure, situated in a fossa on the visceral aspect of the liver. It has a blind end or fundus, a body which narrows to form an infundibulum with an indefinite pouch on the posteromedial surface, and a narrow neck continuous with the cystic duct. The fundus is rounded and usually projects beyond the inferior margin of the liver. It comes in contact with the anterior abdominal wall at the level of the tip of the 9th costal cartilage and outer border of the right rectus muscle.



(Fig. 11 12A) Another method of surface marking is to draw a line from the left anterior superior iliac spine through the umbilicus. The gall-bladder is situated at the site where this line intersects the costal margin (Fig. 11 12B).

**Technique.** The gall-bladder becomes palpable only when distended, it is then felt as a pear-shaped cystic mass. Tenderness over the gall-bladder area is far more common, and is usually indicative of some disease of the organ. There are several methods available for palpating the gall-bladder. (1) With the patient lying flat on the back and the knees flexed, palpation is carried out below the right costal margin in the midclavicular line. The right hand feels for palpability and tenderness, as the patient takes a deep breath. (2) In the bimanual method, besides using the right hand in front for feeling the gall-bladder, the left hand is placed behind the lower part of the chest. By turning the patient to the left, the gall-bladder can be brought closer to the abdominal wall, thus facilitating palpation and making elicitation of tenderness more certain. (3) A useful method of eliciting gall-bladder tenderness is to place the left hand over the right costal margin with the thumb resting over the gall-bladder area. During deep inspiration the descent of the diaphragm causes the gall-bladder to strike against the examiner's thumb whilst the patient complains of a sharp pain and catches his breath (*positive Murphy's sign*). (4) Another method of palpation is to stand or sit behind the patient who is made to sit up and bend forwards, the examiner's two hands are then placed over the two costal margins anteriorly and the patient is made to take a deep breath or to cough.

Other methods have also been recommended for eliciting gall bladder tenderness. One method is to strike a blow with the ulnar surface of the hand over the right subcostal region, the other is to make the patient lie on his stomach and see if tenderness, which was not there with the patient lying flat on his back, arises in the prone position.

**INVESTIGATION** The following observations are necessary during palpation of the gall-bladder.

(1) *Tenderness* Tenderness of the gall-bladder (*positive Murphy's sign*) is a constant sign of inflammation of the gall-bladder, unless the organ happens to be gangrenous. A carcinomatous gall-bladder may also be tender.

(2) *Size* A palpable gall-bladder usually means a gall-bladder over 6 or 7 cm. in diameter. A thick-walled, chronically infected gall-bladder, with or without gall-stones, cannot become distended as the result of common bile duct obstruction, on the other hand, when obstruction is due to malignancy (say of the pancreas), the gall-bladder is enlarged and palpable (*Courvoisier's law*).

(3) *Shape* An enlarged gall-bladder is usually pear-shaped, in case of cholelithiasis, however, a palpable lump may be sausage-shaped.

(4) *Consistency* A malignant gall-bladder is hard. Grating or crepitus, when detected in a lump on the right side of the abdomen, should suggest the possibility of cholelithiasis, such a finding is however rare.

(5) *Surface* When malignant, an enlarged gall-bladder displays an irregularly nodular surface and is frequently associated with an enlarged and nodular liver

(6) *Fluctuation* This may be demonstrable in case of hydrops of the gall-bladder, a rounded or globular fluctuating mass being palpable in the right upper quadrant of the abdomen

(7) *Movement with respiration* A gall-bladder swelling usually moves downwards during inspiration and upwards during expiration

(8) *Mobility* A gall-bladder is movable from side to side, but not, as a rule, upwards or downwards

(9) *Boas's sign* In acute cholecystitis there may be an area of hyperaesthesia over the right subscapular area

**DIFFERENTIAL DIAGNOSIS** An enlarged gall-bladder may have to be distinguished from

(1) *Posed right kidney* Whilst such a kidney is mobile and difficult to localize, an enlarged gall-bladder is prominent and more constant in position. The kidney, unlike a gall-bladder swelling, can be displaced forwards towards the anterior abdominal wall or downwards towards the pelvis, and is bimanually palpable

(2) *Liver enlargement or tumour* A carcinomatous deposit in the liver may simulate a palpable gall-bladder, however, it is much harder to the feel and associated with other projections or nodules over the liver surface. A gumma, abscess or cyst of the liver, or a Riedel's lobe may occasionally be mistaken for an enlarged gall-bladder

(3) *Pyloric tumour* A tumour of the pylorus has its long axis at right angles to that of the gall-bladder and is firm or hard to the feel, gastric symptoms are usually prominent

(4) *Transverse colon* A swelling of the transverse colon has its long axis perpendicular to that of the gall-bladder. Faecal masses may be felt elsewhere in the colon

(5) *Pancreatic cyst* A pancreatic swelling is usually larger, does not move with respiration, is more centrally placed and displays a band of colonic resonance in front

## PALPATION OF KIDNEYS

**Regional anatomy** The kidneys, on either side of the dorsolumbar spine, lie behind the peritoneum, on the posterior abdominal wall. They are about 4 inches long, 2-1/2 inches wide and 1-1/2 inches thick. The hilus of the kidney usually lies opposite the space between the transverse processes of the first and second lumbar vertebrae. The upper poles are nearer the midline than the lower, and usually correspond to the level of the upper border of the 12th thoracic vertebra, the lower poles are 1 to 2 inches above the iliac crests. The left kidney, which is a little longer and narrower, lies somewhat higher than the right. The 12th rib crosses the middle part of each kidney. The lower pole of the right kidney is on a level with a point 2-1/2 cm above the umbilicus.

The costovertebral angle corresponds to the angle between the last rib and outer border of the erector spinae muscle. In case of renal infection, tenderness on pressure is often encountered in this angle.

**Technique** Palpation of the kidneys may be carried out in one of several ways. (1) Usually, the *bimanual method* of palpation (Fig 11 13) is employed. The left hand is placed behind the costovertebral angle and exerts pressure on the kidney, while the palpating right hand is placed high up in the hypochondrium, the patient is then asked to take a deep breath, and the pulps of the

fingers of the two hands are approximated whilst expiration is in progress. It is advisable to palpate the right kidney from the right of the patient and the left from the left side.

(2) In thin subjects and in children, bimanual palpation can be carried out *with one hand*, the fingers being used to apply pressure in the costovertebral angle whilst the thumb feels the anterior aspect of the kidney or its pole, the relative positions of the fingers and thumb may be reversed if found more convenient.

(3) In case of suspected ptosis of the kidney, it is advantageous to palpate the kidney with the patient turned to the sound side, turning of the patient causes the affected kidney to leave its bed and drop downwards and medially.

(4) The kidney can also be palpated with the patient sitting up, and the examiner standing behind the patient.

Normal kidneys are rarely palpable in patients of normal or hypersthenic habitus. The lower pole of the right kidney may however be palpable in asthenic individuals because of the liver. The left kidney is never palpable unless enlarged or displaced. A ptosed kidney can be recognized by an absence of the organ from its normal site and by the fact that it can be pushed back into normal position.

**INVESTIGATION** During palpation of the kidneys, the following features must be studied.

(1) *Situation* If the midclavicular line is extended to a point 2 finger-breadths below the costal arch, an imaginary perpendicular line projected from that point into the depth of the abdomen will reach the normal kidney. An enlarged kidney or renal mass frequently extends well below the costal margin. A renal tumour is usually situated lower and more laterally than most abdominal masses, and tends to grow forwards within the abdomen so as to fill up the hypochondrium eventually.

(2) *Size* A renal swelling may be small enough to offer diagnostic difficulty or large enough to attract the patient's attention and form a visible lump.

(3) *Shape* The normal kidney is an ovoid structure with bluntly rounded margins.

(4) *Consistency* The normal kidney has a peculiar resilient feel. A renal tumour may be either of normal consistency or hard and nodular, a hydronephrotic kidney may be firm or soft, a polycystic kidney is usually nodular.

(5) *Tenderness* Infection of either the kidney or the perirenal tissue may result in an extreme degree of tenderness on palpation, elicitation of tenderness may however be prevented by severe muscle spasm.

The site of maximum renal tenderness is the angle between the 12th rib and sacrospinalis muscle (renal angle) Tenderness may be elicited on palpation or brought out more sharply by fist percussion over the area

(6) *Movement with respiration* The normal kidney moves up and down with respiration Respiratory movements are usually normal in case of a polycystic kidney, normal or impaired with a hydronephrotic kidney and absent in the case of a malignant kidney, because of fixation to perirenal tissues A congenitally mobile kidney can be bimanually grasped and prevented from slipping back during expiration

(7) *Surface* In polycystic disease, palpation of the kidney surface may reveal multiple grape-like clusters, in hydronephrosis the surface feels smooth and rounded

(8) *Fluctuating* A large, soft and fluctuating mass in the region of the kidney is suggestive of hydronephrosis or pyonephrosis

(9) *Change in size* A soft fluctuating mass which appears and disappears, or tends to increase rapidly in size, from time to time, suggests intermittent distension of the pelvis and calyces with urine

**DIFFERENTIAL DIAGNOSIS** The following conditions may have to be distinguished from a kidney lump

(1) *Retroperitoneal tumour* Such a mass is usually situated in one of the upper abdominal quadrants, does not move with respiration, and is more centrally placed than a renal tumour

(2) *Enlarged spleen* The differential diagnosis between an enlarged spleen and a kidney lump has been already discussed

(3) *Tumour of bowel* A malignant tumour of the colon is usually hard and nodular, may be felt anywhere in the abdomen depending on the site affected, is usually non-tender and mobile, but cannot be pushed behind the costal arch, visible peristalsis may be observed at times

(4) *Pancreatic cyst* This may appear as a mass in the left epigastrium, left hypochondrium or both, rarely, it occupies the right side or the whole of the abdomen The tumour is usually smooth, rounded, deeply seated and may feel firm, hard or fluctuant

(5) *Perinephric abscess* There is a bulging mass in the loin or flank, associated with acute tenderness, oedematous skin and rigidity of the overlying muscles, the thigh and leg on the affected side may be kept flexed

(6) *Pelvic tumour* An ovarian cyst usually occupies a more anterior position, possesses a greater range of mobility, does not slide back into the loin or under the costal margin like a kidney lump, and is associated with a zone of resonance, rather than dullness, in the loin

(7) *Riedel's lobe* This moves synchronously with and to the same extent as the liver during respiration, its upper border merging with the lower border of the liver It is more superficial than the kidney and its sharp and firm lower edge can be easily palpated

## PALPATION OF STOMACH

The two main features to look for, during palpation of the stomach, are tenderness and the presence of a mass

**Tenderness** Superficial tenderness is elicited either by stroking the region of the stomach with a finger nail or by applying digital pressure to the abdominal wall (rendered rigid by making the patient raise his head) Deep tenderness can be detected by deep palpation of the area.

In peptic ulcer, a rather sharply circumscribed area of tenderness is common over the site of the lesion. A small ventral hernia, which may cause a similar persistent and local epigastric tenderness, may be missed, unless the patient is asked to contract his abdominal muscles by raising the head. In a thin or asthenic individual, pressure over the anterior aspect of the vertebral column, in the epigastric region, may cause tenderness in the absence of any local lesion. Deep epigastric tenderness may be elicitable in case of pancreatic disease.

**A palpable mass or lump** Whilst a tumour involving the greater curvature or pylorus is easy to feel with the patient lying flat on his back, the right lateral position is preferable for a satisfactory palpation of a tumour of the body or the upper end of the stomach. A stomach mass may be of the nature of a new growth, usually malignant, or an inflammatory lump.

In the case of *new growth*, the position of the mass varies with the site and size of the tumour, the habitus of the patient and the thickness of the abdominal wall. A tumour of the anterior wall or greater curvature is usually palpable and is more obvious with a full stomach. A growth of the pylorus is frequently palpable even when small. In the pyloric stenosis of infants, a hard and knotty lump may be palpable, between the umbilicus and costal margin, along the outer border of the right rectus muscle, especially during the phase of contraction. A gastric tumour is usually best felt during deep inspiration.

In the case of an *inflammatory mass*, there is usually a tender palpable lump, about the size of a thumb, placed transversely, in the midline above the umbilicus. It is usually caused by thickening and induration of the pyloric segment of the stomach secondary to an ulcer.

#### PALPATION OF PANCREAS

Because of its deep position within the abdomen, the pancreas is rarely palpable unless greatly enlarged. In a markedly visceroptotic subject, a normal pancreas may occasionally be palpable.

**Technique.** A special method of palpation, recommended for the pancreas, is to make the patient lie on his back on a firm surface, with the legs flexed. The examiner, standing on his right, places the right hand, with the fingers slightly flexed, over the abdomen, along the lateral margin of the left rectus muscle. During deep inspiration, the fingers of the right hand, reinforced by pressure from the fingers of the left hand, are gradually pushed into the abdominal cavity in the direction of the left border of the vertebral column, whilst with the palm of the right hand the rectus is pushed towards the midline.



Fig 11 11 Surface marking of the liver

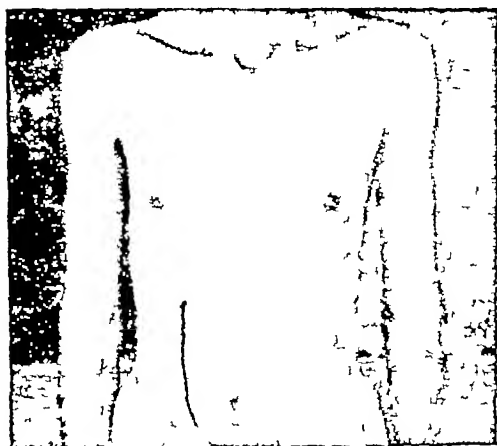


Fig. 11 12A

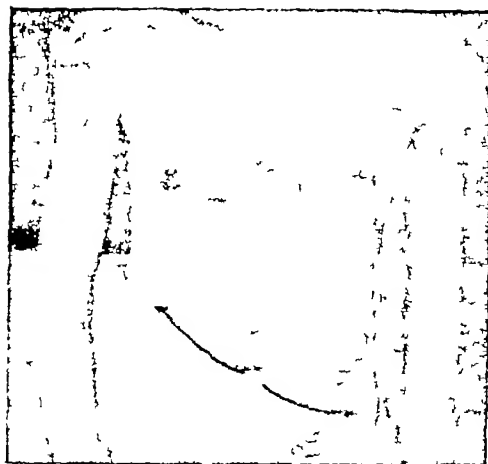


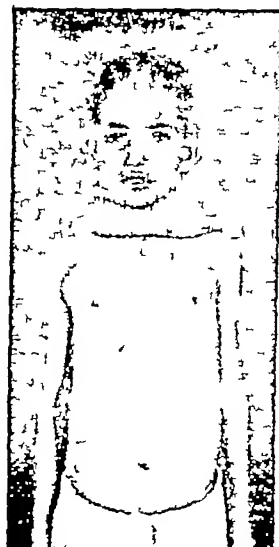
Fig 11 12 B

Surface marking of gall bladder



Fig 11 13 Bimanual palpation of kidney

Fig 11 14 Ascites Prominence of hypogastrium and bulging of flanks Note enlarged lymphnodes in the neck and the right axilla A case of tuberculous peritonitis



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Fig 11 11 Surface marking of the liver



Fig 11 12A

Surface marking of gall bladder

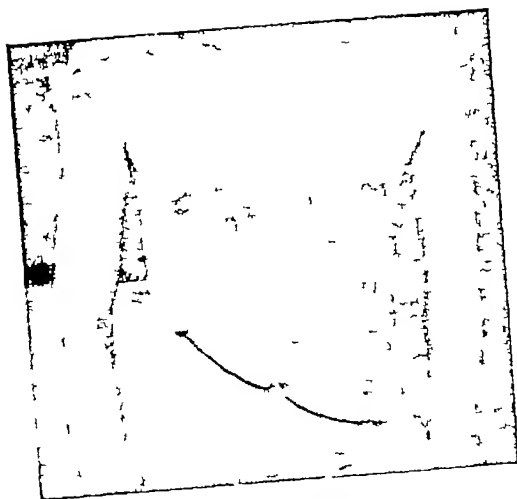


Fig 11 12 B



Fig 11 13 Bimanual palpation of kidney

Fig 11 14 Ascites Prominence of hypogastrium and bulging of flanks Note enlarged lymphnodes in the neck and the right axilla A case of tuberculous peritonitis







*Characteristics* A pancreatic lump, usually caused by a tumour or cyst, when palpable, presents itself as an ill-defined mass in the epigastric and upper umbilical regions. It shows little or no movement during respiration, is slightly mobile on digital manipulation, frequently transmits the pulsation of the abdominal aorta, and is separated from the liver and spleen by areas of resonance.

#### PALPATION OF SMALL INTESTINE

A palpable intra-abdominal mass involving the small intestine may result from intussusception, tumour, tuberculosis or regional ileitis.

The most revealing physical sign in such a case and indicative of organic obstruction is the presence of a persistently dilated loop or loops of small intestine, associated with visible peristalsis, and at times with a tense, sausage-shaped doughy tumour. The peristalsis may be palpable as an alternate hardening and softening of the loop of bowel. A circumscribed mass, although rare in ulcerative tuberculosis, is commonly associated with the hyperplastic form of intestinal tuberculosis.

#### PALPATION OF COLON

The *pelvic colon* is normally palpable as a freely movable sausage-shaped structure, often in close proximity to the anterior abdominal wall, and about an inch medial to the iliac crest. Its size and consistency vary with the amount of faecal matter contained, and with its degree of tone at the time of examination. When spastic or contracted, the colon is felt as a narrow tube-like structure, at times, hard scybalous masses are palpable. The pelvic colon tends to be tender and spastic during the acute stages of ulcerative colitis and diverticulitis.

The *transverse colon* may be normally felt in thin subjects as a movable, transversely placed tubular structure just above the umbilicus.

A tumour in the region of the *hepatic flexure*, being frequently adherent to the under-surface of the liver, tends to descend during deep inspiration. Assumption of the erect posture may cause a lump of the *ascending colon* to descend by a few inches, and thus render palpable a lump otherwise undetectable in the lying-down position.

The *ileo-caecal region* should be palpated with special care in patients suspected of intestinal tuberculosis, amoebic colitis, ileo-colitis, appendicitis or regional ileitis.

#### URINARY BLADDER

An over-distended bladder may be felt as a somewhat tender and rounded cystic mass above the symphysis pubis and extending upwards even up to the umbilicus.

In a recently observed case, the urinary bladder, through chronic retention, had become massive enough to simulate an ascites, on catheterization, it yielded four gallons of retained urine

### HERNIAL SITES

It is wise to palpate the inguinal and femoral regions, on the two sides routinely for hernia, both at rest and during the act of coughing, whilst clinically examining the abdomen. This is particularly important in the case of obscure lower abdominal pain

### PERCUSSION

**Technique** The technique of percussion in the case of the abdomen is no different from that employed with the chest, except that a lighter stroke is usually preferred in the case of the former

The normal abdomen is tympanitic because the greater part of it is occupied by the stomach and intestines. Except for liver dullness and the occasionally appreciable splenic dullness, a high degree of resonance is usually elicited over the abdomen. Depending on the amount of gas in the stomach and intestines, the normal percussion note over the abdomen may vary considerably. Abnormal dullness on percussion, over one or more regions of the abdomen, may be due to tumour, cyst, enlargement of liver or spleen, enlarged glands, aortic aneurysm, malignant deposits, or to ascites, in which case the dullness tends to be bilaterally symmetrical

**VALUE OF ABDOMINAL PERCUSSION** Percussion of the abdomen is particularly valuable in the detection of meteorism, ascites, enlargement of intra-abdominal organs, and subphrenic abscess

**Meteorism, tympanitis or gaseous distension** An increased area of tympany or exaggeration of the normal tympanitic note over the left hypochondrium suggests excess of gas in the stomach or splenic flexure. Similar signs over the right hypochondrium may be due to gaseous distension of the hepatic flexure. Abnormal tympany in the right upper abdomen is often due to organic bowel obstruction. A high-pitched note is more suggestive of distension of the small intestine than of the stomach or colon. Tympany of the whole abdomen, including the flanks, with marked gaseous distension of the abdomen is referred to as meteorism or tympanitis. It may be due to some serious infection, toxic state, general peritonitis or artificial pneumoperitoneum

**Ascites** A collection of free fluid in the peritoneal cavity is referred to as ascites. Over 1500 ml of fluid must collect in the peritoneal cavity before

its presence can be detected by physical examination, this is because of the large size of the abdominal cavity and its many recesses. A symmetrical bulging of the flanks in the recumbent position or a prominence of the hypogastrium in the erect posture (Figs 11 14, 15, 16) should suggest the possibility of ascites. The following special tests may then be carried out to confirm the presence of free fluid

**FLUID THRILL** This characteristic sign of ascites may be elicited by placing one hand over one flank, whilst with the finger or fingers of the other hand a sharp flick or tap is delivered over the opposite flank (Fig 11 17). By this manoeuvre a fluid wave is initiated in the fluid and conducted to the opposite flank, where it is felt by the palpating hand as a tap or thrust. To avoid a false sensation of thrill, particularly in a person with a fat abdominal wall, either the patient himself or a third person is made to place the ulnar edge of the hand firmly and vertically along the linea alba, thus tends to damp out any vibrations which may be conducted through the abdominal wall itself. A fluid wave is usually demonstrable in case of massive ascites, where the other signs of free fluid are difficult to elicit. A fluid thrill rarely occurs with a large cyst, hydramnios or large hydronephrosis.

**HORSE-SHOE SHAPED DULLNESS** In ascites with a moderate amount of fluid, the distribution of dullness in the lying-down position is confined to the flanks (Fig 11 19A). With a larger amount of fluid, the dullness extends

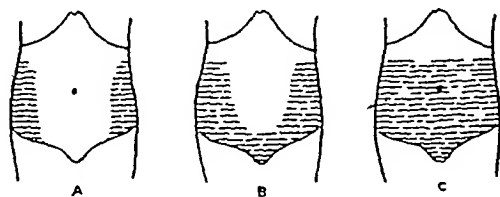


Fig 11 19 Dullness on percussion in ascites. A In flanks with moderate collection. B Horse-shoe shaped with larger amount of fluid. C Total dullness due to massive accumulation of fluid, or adhesions of mesentery preventing floating up of intestines.

from the flanks to the pubic and hypogastric regions because of gravitation of fluid to the most dependent parts, the umbilical and epigastric regions remain resonant because of the floating up of the intestines. Thus, on percussion, the area of dullness appears horse-shoe-shaped with a concave upper border (Fig 11 19B). When the collection of fluid becomes massive, the abdomen as a whole

sounds dull, except for a small area of tympany over the umbilical region (particularly in children) (Fig 11 19C). This may be demonstrated by percussing in various directions from the umbilicus outwards, the para-umbilical zone of tympany ends in a dull note as the flanks are approached. Alternatively, percussion may be carried out horizontally outwards from the midline, towards each flank, the line of dullness being marked out with a skin pencil on either side.

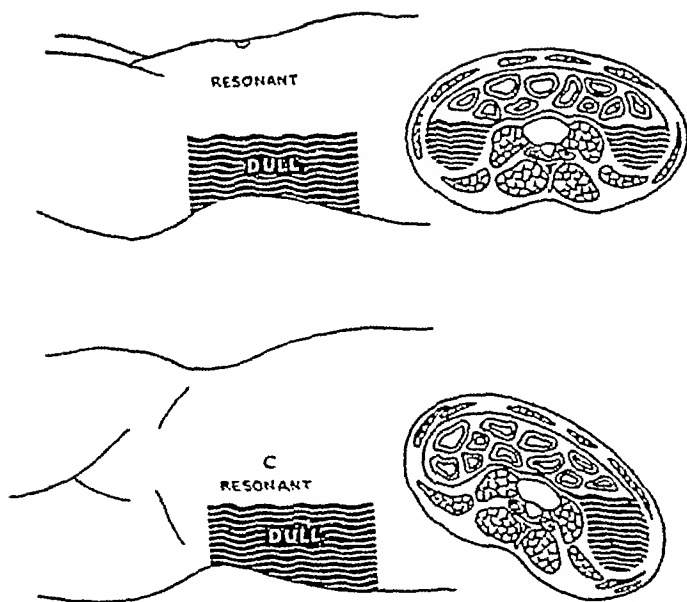


Fig 11 20 Shifting dullness in ascites

**SHIFTING DULLNESS** (Fig 11 20) Turning a patient with ascites to one or other side permits the fluid to gravitate to the dependent parts of the abdomen and the flank which is uppermost to become hyper-resonant, due to the floating up of the air-containing viscera. Demonstration of such a shift of the band or area of dullness from one side to the other with a change in the patient's position, is an important sign of free fluid. It can be demonstrated as follows. The upper border of dullness, in each flank, is determined by percussion. The pleximeter finger is then kept in position on one flank, the patient being asked to turn to the other side. After allowing a few seconds for the intestines to float up, percussion of the pleximeter finger, at the same site, displays a tympanitic instead of the dull note. This is referred to as *disappearing dullness*. At the same time, an increased width of dullness can be demonstrated by percussion of the lower flank, thus proving the *shift* of fluid from one side to the other.

Another method of demonstrating a fluid-shift is to turn the patient slightly to the right side and then to percuss the right flank with the pleximeter finger applied rather firmly over a definitely dull area. The finger is then pressed more and more deeply in the abdominal wall and percussion continued. When the fluid is displaced, the abdominal wall comes against the caecum and the note becomes resonant, when the pressure is released, the dull note returns.

Failure of fluid to shift with change of position, in spite of ascites, may be due to (a) an inability on the part of the intestines to float up because of adhe-

sions, as in tuberculous peritonitis, or to (b) massive ascites, the collection of fluid may be large enough to prevent contact of the bowel with the abdominal wall

Shifting dullness may rarely be noted in the absence of ascites, as in case of a large cystic swelling within the abdomen, or with an excessive amount of fluid within the intestine, the fluid contents may shift sufficiently, with change of position, to alter the note on percussion

**PARA-UMBILICAL ZONE OF DULLNESS** For detecting small amounts of fluid, within the peritoneal sac, not demonstrable by any of the above methods, percussion of the para-umbilical region, after putting the patient in the knee-elbow position, is recommended. The area around the umbilicus, which is normally tympanitic, is dull on percussion if it contains fluid

In women, an early or mild degree of ascites can be demonstrated by palpating for fluid through the pouch of Douglas during vaginal examination

The abdomen must always be examined after removal of ascitic fluid. Tumours or enlargements of organs, previously obscured by the fluid, may become apparent after paracentesis

**The puddle sign** This sign is said to be of aid in the diagnosis of minimal (as little as 120 ml) ascites. To elicit the sign, the patient lies in the prone position for five minutes, after which he goes on "all fours" so that the middle portion of the abdomen is dependent. One flank is then percussed by repeated light flicking at a constant intensity and the diaphragm chestpiece of the stethoscope placed on the most dependent portion of the abdomen where the fluid puddle collects. The chestpiece is then gradually moved to the flank opposite the percussion note. A marked change in the intensity and character of the percussion note, as the stethoscope is moved towards the opposite flank, denotes the presence of fluid. The patient then sits while the examiner holds the stethoscope on what was the most dependent part of the abdomen. A loud and clear percussion note in this position confirms the initial finding.

**Penfield method** Patient is made to sit up in bed upright. This makes the intestines float upwards. One hand is now used to percuss directly over one of the quadratus muscles. This sets up a succession wave which can be felt by keeping the other hand on the anterior abdominal wall if fluid is present

#### Causes of ascites

##### A *Conditions in which ascites is the main feature*

(1) Diseases of peritoneum Tuberculous peritonitis, spontaneous bacterial peritonitis, malignant peritonitis

(2) Portal vein obstruction Cirrhosis of liver, thrombosis of portal vein, enlarged portal lymphnodes

(3) Inferior vena caval obstruction Adherent mediastino-pericarditis, mediastinal new growth, thrombosis of vein

(4) Diseases of female reproductive organs Ovarian tumour, Meig's syndrome (ovarian fibroma, ascites and pleural effusion)

(5) Diseases of lymphatic system Injury to or obstruction of the thoracic duct

(6) Pancreatic ascites

(7) Rupture of a hollow viscus

**B Ascites associated with generalized anasarca**

- (1) Cardiac diseases Congestive cardiac failure
- (2) Kidney diseases Nephrosis
- (3) Hypoalbuminaemia Nutritional deficiency states, protein losing enteropathy
- (4) Polyserositis
- (5) Epidemic dropsy

**Mechanism of ascites** The mechanism of production of ascites depends greatly on the aetiological factor or factors present, more than one mechanism may be involved

(1) **INFLAMMATION** Irritation of the peritoneum by inflammation leads to increased permeability of its capillaries and transudation of fluid into the peritoneal cavity, also, re-absorption of the intraperitoneal fluid is reduced, partly as the result of oedema of the peritoneum and partly because of the high protein content of the exudate

(2) **VENOUS OBSTRUCTION** In inferior vena caval obstruction, ascites is due to mechanical obstruction In portal venous hypertension due to cirrhosis, several factors are involved in the production of ascites (a) lowering of colloid osmotic pressure due to diminished serum albumin, (b) increased sodium and fluid retention due to increased aldosterone secretion, (c) accumulation of oestrogens which are slowly excreted in liver disease, (d) reduction in liver blood-flow and filtration rate in some cases, and (e) high pressure in portal circulation localizing the fluid retention to the abdominal cavity and engorging the liver so that excess hepatic lymph is produced

(3) **LYMPHATIC OBSTRUCTION** Involvement of the mesenteric lymphatics, receptaculum chyli or thoracic duct leads to chylous ascites, as a result of obstruction to the flow of lymph

(4) **RUPTURED VISCUS** Rupture of an intra abdominal organ, by causing an out-pouring of blood, cystic fluid or contaminated material, results in a collection of fluid within the peritoneal cavity

**Differential diagnosis of ascites** The following conditions have to be distinguished from ascites

(1) **OBESITY** The protrusion of the abdomen in obesity is symmetrical and out of proportion to the rest of the body, the navel is depressed and the transverse cutaneous folds of the abdominal wall exaggerated In extreme obesity, the fat may hang in front like an apron (Fig. 11.18) with protrusion of the flanks and supra pubic region In obesity, there is no shifting dullness or fluid wave

(2) **GASEOUS DISTENSION** There is no bulging of the flanks, the abdomen is tympanitic all over, and a ladder-like pattern may be obvious over the abdomen on inspection

(3) **PREGNANCY** The gravid uterus usually causes a symmetrical bulging of the abdomen, with the anteroposterior diameter of the abdomen markedly increased, and the maximum girth of the abdomen below the umbilicus In ascites, on the other hand, the maximal bulging is in the flanks and the maximum girth above the umbilicus In pregnancy, the dullness on percussion is centrally located, with tympany in the flanks Changes in posture produce little or no change in note

(4) **OVARIAN CYST (Fig. 11.21)** In the early stages, the enlargement of the abdomen may be noticeable more on one side. The umbilicus is nearer to the ensiform cartilage than the pubes unlike ascites where it is nearer to the pubes A fluid thrill may be obtained only in front (and not far out in the flanks) The upper border of dullness in front is convex above, whereas in ascites it is horse-shoe shaped and with concavity above. The outline of the upper border of the cyst may be visible during respiratory movement The maximum girth of the abdomen is below the umbilicus, and the distance between the umbilicus and the anterior superior iliac spines on the two sides unequal



Fig. 11 15 Ascites in cirrhosis of liver Note gynecomastia, scanty body hair, prominent anterior abdominal veins and epigastric hernia The umbilicus is everted



Fig 11 16 Malignant ascites Ascites due to malignant infiltration of peritoneum secondary to a primary carcinoma in the left breast

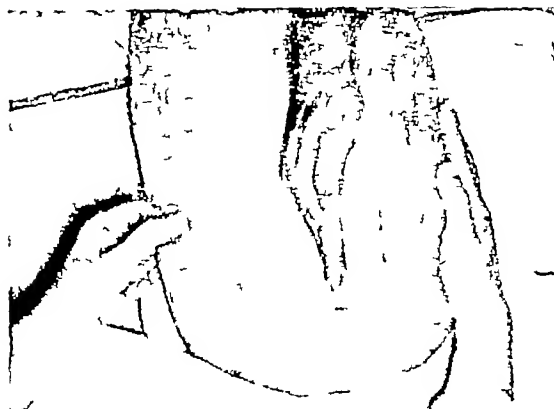


Fig 11 17 Method of eliciting fluid thrill

Figs 11 19 and 11 20, see pp 395, 396 respectively



Fig 11 18 Obesity simulating ascites





Fig 11 22 Megacolon causing protuberant abdomen

Fig 11 21 Distension of abdomen due to ovarian cyst



Prot belly of rickets



Fig. 11 24 Enormous and almost uniform distension of abdomen due to massive splenomegaly in chronic myeloid leukaemia

(5) **MEGACOLON** (Fig 11 22) The abdominal enlargement may be extreme, with the girth and distance between the xiphoid and umbilicus markedly increased. The enlargement, which is symmetrical, is frequently associated with diastasis of the recti muscles, visible intestinal peristalsis and prominence of the superficial abdominal veins. Flabbiness and atrophy of the muscles of the extremities and loss of subcutaneous fat throughout the body may be associated with the abdominal enlargement.

(6) **VISCEROPTOSIS** In generalized visceroptosis, the abdominal wall has a tendency to bulge laterally or in the flanks in the recumbent posture, and in the hypogastric region in the upright posture. Divarication of the recti and visible systolic pulsation of the abdominal aorta may be noted in the epigastrium.

(7) **LORDOSIS** An extreme degree of lordosis may simulate abdominal distension by causing protrusion of the lower abdomen.

(8) **RICKETS IN CHILDHOOD** (Fig 11 23) The pot-belly of rickets, which is due to weakness or atrophy of the abdominal muscles, tympanitic distension and enlargement of the liver and spleen, may be mistaken by the unwary for an ascites.

(9) **ENORMOUS ENLARGEMENT OF SPLEEN** In cases of considerable enlargement of the spleen, the abdomen may appear to be uniformly distended as though due to ascites (Fig 11 24).

(10) **OVERDISTENDED BLADDER** A massive overdistension of the urinary bladder may simulate an ascites, particularly when it extends to above the level of the umbilicus. In the case of the overdistended bladder, however, the mass is usually firm and rounded, palpation causes pain or discomfort, the flanks are tympanitic, and shifting dullness is not obtained on change of posture.

**Size of liver** Percussion may prove of value in determining the exact size, shape and degree of enlargement of the liver and is the only method of determining a small liver. To outline the upper border of the liver, it is customary to percuss downwards from the second right rib, along the mid-clavicular line and using a fairly heavy percussion stroke, until a definite impairment of note is noted. Normally, the upper border is to some extent dependent on the habitus of the patient. It is usually at the level of the fifth rib in hypersthenic, behind the fifth interspace in normosthenic, and behind the sixth, seventh or eighth rib in asthenic individuals. It may alter its position by one or two interspaces during deep inspiration. The anterior hepatic projection can be obtained by measuring the vertical distance between the uppermost and lowermost points of hepatic dullness by percussion in the right mid-clavicular and midsternal lines (Normal 8-12 cm). When the lower edge of the liver is palpable, the position of the upper border of liver dullness may serve to differentiate ptosis of the liver from enlargement. An upward extension of liver dullness may be caused by a liver abscess, basal consolidation of lung, or subphrenic abscess, which not only elevates the diaphragm but causes downward displacement of the liver.

The *lower border* of liver dullness is elicited by percussing upwards, from the umbilicus to the right costal margin or xiphoid, employing a very light stroke until an impaired note is elicited.

**OBLITERATION OF LIVER DULLNESS** When liver dullness is absent or replaced by tympany, either anteriorly or in the mid-axillary line, the condition

is highly suggestive of free air in the peritoneal cavity, as in the case of perforation of a peptic ulcer, with the rare exception, of course, of an extreme degree of pulmonary emphysema or marked distension of the bowel

**DECREASE IN SIZE OF THE LIVER.** Although some maintain that there is no method of physical examination which allows one to conclude that the liver is smaller than normal, others are of the opinion that percussion of the upper and lower borders is helpful in determining a diminution of liver size. It is necessary, however, to ascertain that diminution of normal hepatic dullness is not due to a voluminous lung as in emphysema or to a distended colon. If day to day percussion reveals a gradual recession of the upper and lower borders of hepatic dullness, a shrinking or atrophy of the liver may be presumed.

**HYDATID THRILL.** In case of suspected hydatid cyst of the liver, when the middle finger of the left hand is placed over the lump and percussed, a peculiar quivering vibration of the finger is experienced. Although considered pathognomonic by some, the value of this sign (hydatid thrill) in diagnosis is questionable.

**PERCUSSION OR SHOCK TENDERNESS OF LIVER.** Tenderness or pain elicited by firm pressure or percussion, along an intercostal space over the right lower chest posteriorly, is an important sign of hepatic inflammation. In infective hepatitis, even in patients with no palpable enlargement of the liver, heavy percussion below the angle of the right scapula may cause severe discomfort.

**Size of the spleen.** Increase in size of the spleen can be roughly determined by percussion as in the case of the liver. There are two methods. (a) The patient is placed in the right lateral recumbent position with the left arm extended forward and upward, so as to clear the left lower part of the thorax. In this position, the spleen lies above both the stomach and the colon. Percussion is begun at the lower level of pulmonary resonance, in the posterior axillary line, and continued obliquely downwards towards the lower mid-anterior costal margin. Normally, the upper border of splenic dullness extends for about 6 to 8 cm above the costal margin. Increase of dullness to more than 8 cm in the adult indicates splenic enlargement. (b) With patient in supine position percussion in the 8th or 9th interspace in the anterior axillary line gives a resonant note if the spleen is normal in size, and the resonance persists while the patient takes a deep breath. With enlargement of the spleen the percussion note in the same spaces is dull on full inspiration as well as full expiration.

**Gall-bladder.** An enlarged gall-bladder is usually dull to percussion. Being directly behind the parietal peritoneum, the colon rarely interferes with its percussion. Gall-bladder dullness is continuous with that of the liver.

**Kidneys** The kidney borders can often be outlined by percussing posteriorly, starting at or about the ninth rib close to the spine. Renal dullness is usually elicited in the tenth interspace. Presence of tympany in that area indicates absence of the kidney on that side.

Occasionally, a markedly enlarged kidney may not be felt by palpation, particularly if it happens to be soft, as in the case of hydronephrosis. Such a mass can be readily outlined as a rule by percussion. Percussion is of particular value for outlining an enlarging mass in the flank, following renal trauma, such a sign is indicative of progressive haemorrhage.

*Fist percussion of the kidney* Deep tenderness over the kidney region, as the result of a renal lesion, may be elicited at times by striking the region of the costovertebral angle with the closed fist.

**Urinary bladder** A distended urinary bladder can be made out by percussion. There is a zone of dullness in the suprapubic region, extending up to the upper border of the distended bladder, associated with tenderness on percussion.

**Pelvic tumour** Percussional dullness over the suprapubic region may be due to a pregnant uterus, a large uterine or ovarian tumour or an ovarian cyst.

**Size of the stomach.** The approximate size of the stomach can be assessed by percussion, if the viscus contains mainly air or gas. When direct percussion is carried out from a point slightly to the left of the midline of the lower abdomen, obliquely, towards the ensiform cartilage, a splash is detected when the lower border of the stomach is reached, when not heard, the patient is given a glass of water to drink, and the procedure repeated. Besides being of value in ascertaining the position of the lower border of the stomach, percussion may reveal a delay in emptying of the stomach. If a stomach splash persists for more than four hours after a full meal or two hours after a glass of milk or water, a delayed emptying of the stomach should be suspected.

**Obliteration of Traube's area.** The size of Traube's area of stomach tympany depends to some extent on the character and amount of stomach contents. Its obliteration may be due to a left-sided pleural effusion, enlargement of the left lobe of the liver or less often to a moderate or marked enlargement of the spleen.

**Subphrenic abscess** When gas is present below the diaphragm in case of subphrenic abscess, percussion may reveal a circumscribed area of tympany.

**Resistance of abdominal wall** Direct percussion may be employed at times to determine the degree of resistance of different regions of the abdominal wall.

## AUSCULTATION

Auscultation is an important part of abdominal examination. It is best carried out in deep expiration and with light application of the bell chest piece.

Routine auscultation over all the four abdominal quadrants may give clues to the following.

**I. Peristaltic sounds (Bowel sounds).** NORMAL PERISTALTIC SOUNDS OR BORBORYGMI. These are normally heard over the abdomen and are noises caused by the peristaltic action of the bowel wall on the fluid and gas contents within; they are more marked with an empty bowel.

**EXAGGERATED PERISTALTIC SOUNDS.** Abnormally loud borborygmic sounds may be heard over an area of partial obstruction and are due to the powerful peristaltic action of the hypertrophied muscle coat of the bowel wall trying to eject the contents of the bowel through the narrow or constricted area. Similar noises may also be associated with longer empty bowel purgation, carbonylamine dyspepsia, asphyxia, cyanosis or congestive cardiac failure.

**ABSENCE OF PERISTALSIS (Silent abdomen).** In intestinal atony or paralysis, the peristaltic sounds are apt to be diminished in intensity and occur infrequently or not at all. Local absence of sounds in the right iliac fossa is often a useful sign of localized peritonitis associated with appendicitis.

## II Vascular sounds.

(a) **Murmurs.** *Prone* (prone)—abdominal murmurs are not uncommon in normal healthy individuals young individuals; their frequency is greater in patients with hypertension. *Prone* (prone)—(a) **Renal artery stenosis.** In stenosis of the renal artery a murmur may be heard most clearly just above the umbilicus close to the midline, usually to the left of the midline corresponding to the position of the aorta. It is a high-pitched murmur that may be continuous or to-and-fro with systolic accentuation. To hear the murmur best the patient must be lying on the back raised with his knees flexed, and the examiner should listen as the patient exhales. (b) An upper abdominal murmur may supply the clue due to mesenteric artery stenosis. (c) A murmur at the aortic bifurcation may indicate thrombotic obstruction of the terminal aorta. (d) Systolic bruits can be heard over many aneurysms. (e) Splenic murmurs have been described in the left hypochondrium in association with a tortuous splenic artery, hypertension in case of splenomegaly and rarely in carcinoma of the body of the pancreas. (f) Murmurs over the liver may indicate primary liver cancer, acute alcoholic hepatitis, A-V malformations, or enlarged common hepatic arteries in diabetes.

(c) **Venous hums.** (i) **Right-lygal**—Although less common than innocent arterial murmurs, venous hums arising from the inferior vena cava may be heard. A souffle over the lower abdomen is normal during pregnancy. (ii) **Right-lygal**—(a) A venous hum may be heard usually in the region of the umbilical process or umbilicus in carbonylamine. It is continuous but tends to be louder during systole, in inspiration and when the patient is standing. The hum results from the opening up of the umbilical vein due to portal hypertension. Disappearance of the hum suggests thrombosis of the portal vein. One should suspect Cruveilhier-Benjamin syndrome when one hears a venous hum in a patient with carbonylamine and there is evidence of portal hypertension, splenomegaly and prominent collateral umbilical veins. The Cruveilhier-Benjamin murmur is often intensified by making the patient raise his head from the pillow and usually disappears when pressure is applied immediately above the umbilicus. A palpable thrill over the area where the murmur is louder is common. (b) A venous hum

may be heard occasionally over large collaterals such as the inferior mesenteric vein or after porta-caval anastomosis (iii) Arterio-venous malformations or fistulas within the abdomen give rise to continuous low-pitched murmurs (iv) Aneurysms, narrowing or obstruction of the aorta or its branches may produce continuous abdominal murmurs (v) A venous hum commonly occurs over vascular tumours (metastatic chorio-epithelioma), or over any site where veins are compressed by tumours or lymphnodes

### III Friction sounds.

(a) *Over the liver*—Friction may be audible (and palpable) in the right hypochondrium usually due to recent liver biopsy, carcinoma of the liver and perihepatitis or liver abscess (b) *Over the spleen*—A rub may be heard after splenic venography, or over an area of splenic infarction, or surface tumour

### IV Miscellaneous sounds

**Succussion sounds** (Gastro-intestinal splash) A splashing sound, due to the presence of both fluid and gas in the stomach, may at times be elicited by shaking the patient The splash is particularly pronounced in case of atonic or dilated stomach or intestine

**Size of stomach** Auscultation may be employed for roughly gauging the size of the stomach or for detecting gastric dilatation The chestpiece of the stethoscope being placed over the centre of the stomach, the skin of the abdomen is scratched with a finger nail along various lines radiating from the centre A sudden change in intensity or character of the scratching sound is noted along each line By joining these various points, the outline of the stomach can be roughly delineated A tuning fork may be substituted for direct percussion with the finger

**Oesophageal sounds** In oesophageal obstruction, for example from carcinoma, auscultation over the lower part of the back, along the spine, may give an indication of delay in the passage of fluid from the pharynx to the cardiac end of the stomach The patient in the upright position holds some fluid in the mouth, until instructed to swallow, the examiner listens just below the xiphoid for two sounds, first the sound of fluid entering the oesophagus from the pharynx, and secondly the sound of its passage through the cardiac sphincter Between the two sounds, there is usually an interval of 5 to 10 seconds, a considerable interval between the sounds or an absence of the second sound is suggestive of oesophageal obstruction In obstruction of the lower oesophagus, instead of one loud squirt, only a faint intermittent tinkling may be heard for 15 seconds to several minutes after the act of deglutition

**Abdominal mensuration** A periodic measurement of the abdomen may be of value in following up the progress of a case of ascites or of distension of the abdomen from splenomegaly, hepatomegaly or large tumour It may also aid the differentiation of an ascites from an ovarian cyst The *abdominal girth* is usually measured at the level of the umbilicus in front and the tip of the third lumbar vertebra behind Variations in girth from day to day, if any, should be noted carefully

The distance of the umbilicus from the anterior superior iliac spine (on either side) and from the ensiform cartilage and pubes must be measured carefully, whilst in ascites the distance of the umbilicus from the ensiform cartilage is shorter than that between the umbilicus and pubes, in ovarian cyst the reverse generally holds true Also, whilst in ascites the distance between

the umbilicus and the anterior superior spine on either side is the same, in ovarian cyst the measurements are unequal. In ascites the greatest circumference of the abdomen is generally at the umbilicus, whereas in ovarian cyst it is below the umbilicus.

## RECTAL EXAMINATION

Examination of the rectum is of the utmost diagnostic value and should be routinely practised during physical examination of the patient. In the presence of abdominal symptoms, such as constipation, diarrhoea, bleeding per rectum, lower abdominal pain, wasting, loss of appetite, urinary or pelvic discomfort, a rectal examination may prove indispensable.

**Technique** The patient may be made to lie either on his left side with the thighs flexed, or in the knee-elbow position, the latter being particularly suitable for palpation of the prostate gland. A dorsal position with the right leg flexed is convenient for examination of the rectum, when the patient is suspected of having an acute intra-abdominal condition, which does not permit him to adopt any other position but the dorsal.

The perianal skin must always be observed first, for evidence of fistula, piles, abscess or pruritus ani. The patient is then asked to strain down in order to reveal the presence of internal haemorrhoids, which may slowly protrude out, when present.

The lubricated index finger, kept straight, is introduced very gently and without pushing into the rectum, the resistance offered by the anal sphincter being noted. Undue tenderness during the procedure is suggestive of ulcer or fissure within the anal canal, or of haemorrhoid, fistula, abscess or inflamed and hypertrophied papilla.

The size of the rectal ampulla is noted. Abnormal dilatation is common in the rectal type of constipation. In chronic ulcerative colitis, the rectum may be narrowed and the mucosa rough. In lymphogranuloma venereum, a stricture may be felt close to the internal sphincter. In carcinoma of the rectum, a hard and irregular surface or mass may be palpable.

A carcinomatous infiltration of the floor of the pelvis should be carefully looked for, by palpating the anterior wall of the rectum, above the level of the prostate or cervix uteri. Such a finding is suggestive of abdominal carcinoma, particularly affecting the stomach. In acute appendicitis, when the appendix is low in position, tenderness and at times a tense lump may be felt in the cul-de-sac to the right of the recto-sigmoid.

# 12

## Psychiatric Diagnosis

A TRAINING in the clinical diagnosis of psychological ailments is as important as in the rest of medicine. Lack of adequate theoretical and practical training, along with traditional prejudice and stigma attached to psychiatry, has been the source of innumerable fantastic notions amongst medical men about mental illnesses and mental patients. A commonsense but traditional approach to these illnesses, by otherwise scientifically trained doctors, frequently results in a performance no better than that of a quack. Such a situation is hardly desirable and must be corrected as soon as possible. Some teaching institutions also fail to keep pace with the progress of modern psychiatry. It is therefore in the interest of the student to study this branch of medicine well, in order to make his practice of medicine more scientific and rational.

The importance of this speciality lies not only in being able to recognize classical psychiatric ailments, such as the psychoses and neuroses, but also in diagnosing and handling so-called "functional" ailments, which constitute large proportion of the various illnesses, which any physician is called upon to diagnose and treat.

It is now-a-days well recognized that it is the "man" as a whole and *not* the diseased "organ" only that is in need of treatment. The "man" is made up of the "body" and the "mind", the two components being inseparable and having continuous interaction on each other. The incidence of physical symptoms and signs of psychogenic origin is much higher than is commonly believed. No physical symptom can be regarded as adequately studied or treated, unless a simultaneous study of the psychological aspect of the case is also conducted. Not only for psychiatric and psychosomatic illnesses, but also for physical illnesses, psychological evaluation of the *personality* of the individual is necessary. This can be systematically and easily accomplished by medical men, provided they are well trained in the clinical methods of psychological medicine.

The prevalent belief, that psychiatric symptomatology and examination are vague, unreliable and unscientific, is far from true and entertained only by the ignorant and uninitiated.



## NORMAL VERSUS ABNORMAL

It is commonly believed that to a psychiatrist all forms of behaviour are abnormal. This is as untrue as saying that to a physician all human bodies are abnormal. This erroneous notion arises from too narrow and rigid a concept of normality. Differentiating "normal" from "abnormal" is often a difficult task, requiring considerable skill and experience. Normality is a range and not a point. Abnormality in psychiatry implies an exaggeration or deviation from normal, more often of degree than of kind, and resulting from internal or external stress. Ability to stand stress varies with each individual and depends on his "total" constitution, the result of genetic, organic and psychosocial influences. Symptoms in psychiatry often serve a defensive purpose, like fever and inflammation in general medicine. The absence or presence of symptoms and their intensity are determined by the "resistance" of the individual concerned and the degree, type and suddenness of the "stress" imposed.

Apart from genetic and organic factors, it is not easy to define "normality". The prevalent concept that "absence of disease" means normal health is no longer tenable. One must think in terms of "positive health". A normal person should not only be free of all psychotic, neurotic, psychosomatic and behavioral disturbances, but must be also subjectively comfortable, happy and free of disabling conflicts. He should be able to face the normal stresses and strains of everyday life in a realistic way, without getting distressed or disabled. Significant deviations from such an "ideal" state constitute "abnormality" in psychiatric practice.

The pattern of dealing with or combating "stress" determines the "personality" of the individual. This in turn determines his response to future stress, which may or may not result in symptoms. A particular type of response to stress frequently leads to a certain type of personality, which although not strictly speaking abnormal may be significantly different from normal.

There are certain well-defined *types* of "personality patterns", which may lead at times to interpersonal difficulties and social stress. They may also form the substratum for future neurotic, psychotic and psychosomatic disturbances. Hence a knowledge of such personality disorders is clinically useful and important. The following are the main varieties of personality disorders.

## PERSONALITY DISORDERS

*Cyclothymic personality* This is characterized by marked swings of "mood" from exultation to depression. Although more so than in an average person, these swings are not gross enough to constitute a disease-state. The Cyclothymic trait is probably genetically determined and predisposes the individual to manic-depressive psychosis.

A *hypomaniac* individual is a cheerful, optimistic, confident and easy-going person. He not only enjoys life but radiates his cheerfulness to others. He may become aggressive or interfering, and at times a nuisance in serious business. Hypomanics may become professional jokers or clowns. Further elation of the hypomaniac mood may lead to mania.

The *melancholic* individual is a pessimistic, gloomy, lonely individual, who finds life dull and depressing. He is a poor and boring companion. Apart from genetic factors, the melancholic trait is frequently a reaction against "repressed aggression". Deepening of the melancholic mood may result in depressive psychosis.

*Schizoid* personality. The individual is antisocial, poor mixer, sensitive and cold, who prefers day-dreaming and loneliness. He usually selects a job or career, where he has little or no dealing with other human beings. Apart from genetic factors, a highly authoritative or dominant father plays an important role in its causation. Such individuals are prone to schizophrenic psychosis.

An *obsessional* individual is excessively keen on cleanliness, method, system, punctuality and orderliness. He is very persistent, fastidious and a perfectionist. He is usually hard-working, conscientious and has an exaggerated sense of duty. He is rigid, unable to relax and prone to worries. Under stress, he is likely to develop an anxiety state or obsessive compulsive neurosis. Some of these individuals develop paranoid schizophrenia. Roots of this personality, according to Freudian psychopathology, lie in fixation at the "anal" stage of psychosexual development. Hence the condition is sometimes referred to as an "anal personality".

A *paranoid* personality is dominated by suspicions and feelings of persecution and injustice. The individual finds it difficult to trust people and blames others for his own misfortunes. In doing this, he tends to project his repressed hostility, arising out of frustrations and failures, on to the environment. Such an individual is prone to paranoid psychosis.

*Hysterical* personality. The subject is an immature, impulsive, unstable and egocentric person, with a low threshold for stress. Unable to stand any frustration, she is likely to get into fits of temper or hysterics. They love to be the centre of attraction, and exhibit talents and body. Some of these individuals have high dramatic talent and make good actors and actresses. Under stress, they have a tendency to develop hysterical symptoms and signs.

*Psychopathic* personality. Such an individual is immature, impulsive and unstable. He is unable to postpone gratifications and is dominated by anti-social impulses and behaviour, indulging quite frequently in lying, stealing, bootlegging and other criminal activities. He gets a kick out of his antisocial behaviour. The roots of such a personality usually lie in an unhappy or

broken home, with a highly authoritative, cold and aggressive father. The latter provokes rebelliousness in the child, who learns to hate all forms of authority later in life.

*Passive aggressive personality* The individual may be passively dependant or passively aggressive. The *passive dependant* type is totally meek, dependant and submissive, unable to express any aggression although full of it. The *passive aggressive* type expresses aggressiveness in a passive manner in the form of stubbornness, obstinacy, obstructiveness or inefficiency.

*Aggressive personality* Such an individual reacts with irritability, temper tantrums or destructive behaviour to frustrations. Aggressive individuals are full of drive and frequently appoint themselves as leaders. They can become hostile, provocative, antagonistic, overtly aggressive or violent when contradicted or obstructed.

*Inadequate* individuals are usually parasites or chronic drifters. Unable to stand on their legs, they have to lean on others for support. They are timid, ineffective, dependant and miserable, and tend to develop anxiety states or schizophrenia under stress.

## SYMPTOMATOLOGY

The *causes* of mental illnesses are complex and not well understood. Genetic, organic and psychological factors play their parts in varying degrees in different illnesses. Although causation of such an illness may not be clear, individual symptoms of the disease belong to the "psychic life" of the individual. They usually arise from the life experiences of the patient and usually have some meaning or reason, although seemingly meaningless, bizarre or funny. They are the symbolic expressions of defence against repressed images, ideas, feelings and anxieties. They are frequently tension-reducing devices or defences (not unlike fever and inflammation) which act as crutches to the patient. The first line of defence against *anxiety* are mental mechanisms, such as repression, reaction formation, symbolization, projection and rationalization, the excessive and habitual use of which results in various disorders of personality. When this line proves inadequate, symptom formation (neurotic, psychotic or psychosomatic) takes place. For convenience, the human "mind" can be considered in terms of three segments or faculties, viz. (1) Conation (tendency to action), (2) Cognition (intellectual function), and (3) Affection (feeling tones). These are the three components of the mind, the disorders of which merit attention.

## DISTURBANCES OF BEHAVIOUR

Increased activity, decreased activity, repetitious activity, automatic behaviour, negativism and compulsions are some of the important disturbances of behaviour.

*Increased activity* Increased psychomotor activity is commonly seen in mania. The patient is continuously busy and "on the go," but achieves little or nothing. This is usually associated with a flight of ideas. The patient either keeps on talking or doing something or other continuously, without any sense of exhaustion.

*Decreased activity* Decreased psychomotor activity is observed in states of depression. It is typically associated with a reduced pressure of speech and often with mutism. In extreme cases, the patient may remain in a state of stupor.

*Repetitious activities* Persistent and constant repetition of certain movements, known as *stereotypy*, is commonly observed in schizophrenia and compulsions. Stereotypy may be of speech, position or movements of the body.

By *catalepsy* is meant a constantly maintained immobile position of the body. When "*cerea flexibilitas*" or waxy flexibility is present, the limbs and body of the patient can be manipulated at will and are kept in such positions for long periods of time. This condition is commonly associated with catatonic schizophrenia, when the patient is in a state of stupor or semi-stupor.

*Mannerisms* are stereotyped movements of the nature of grimaces, gestures, peculiarities of gait, etc. commonly found in schizophrenia.

*Verbiageration* is stereotypy of speech, with repetitions of meaningless words, phrases and sentences.

*Automatic behaviour*, in the form of *automatic obedience*, is a condition in which the patient automatically or compulsively acts out suggestions and requests. *Echolalia* or imitation of words and *echopraxia* or imitation of movements are also forms of automatic behaviour.

*Negativism* is characterized by behaviour opposite to that suggested or advised. Mutism and refusal of food or of nursing care are some of its various manifestations. In extreme cases, the desired behaviour can only be achieved by suggesting just the opposite. This is common in catatonic schizophrenia.

*Compulsions* A morbid and often irresistible urge to perform an apparently unreasonable act is known as compulsion. The person is aware of the act being irrational, but cannot avoid or control the same. He may, for instance, keep on touching certain objects or walk along cracks or on alternate tiles or count up to 3 or 7 or keep washing the hands or perform complicated rituals. Compulsions are usually associated with obsessive ideas which are acted out. Compulsions frequently serve as "defence reactions" against anxiety and guilt. They are associated with an obsessive compulsive personality and are characteristic of classical obsessive compulsive neurosis. At times, they are associated with a schizophrenic reaction or depression.

## DISORDERS OF PERCEPTION

*Illusions* and *hallucinations* are important disorders of perception. In the case of an *illusion*, there is misinterpretation of a sensory stimulus, for instance a "rope" is mistakenly taken as being a "snake". In the case of a *hallucination*, perception takes place in the absence of a stimulus.

*Illusions*, when associated with clear consciousness, are usually reflections of repressed intense affects, desires, urges and impulses. When associated with clouded consciousness, as in case of toxic-infective states, they have much less psychological significance.

*Hallucinations* may also occur with clear or cloudy consciousness. Those taking place in clear consciousness are usually of psychological significance, in contrast to those associated with cloudy consciousness which are usually secondary to toxic-infective states. In the case of hallucinations, repressed psychological material is projected through one or other of the senses, resulting in auditory, visual, tactile, gustatory or olfactory hallucinations. Rarely, fleeting hallucinations are described by even normal persons, when in a tensely emotional or deprived state. Hypnagogic hallucinations and those associated with LSD and mescaline are normal. Hallucinations are common in acute schizophrenic psychosis and are often associated with delusions.

*Auditory hallucinations* are common in paranoid schizophrenia. The patient may hear pleasant or more often unpleasant, derogatory, obscene or accusing remarks in the form of voices coming from within or without. Hallucinations of a commanding nature may be convincing or compelling enough to result in direct and dangerous actions.

*Visual hallucinations* are less common than auditory ones. They are usually associated with toxic-infective psychosis and with acute, reversible organic brain disorders like delirium.

*Olfactory hallucinations* are frequently associated with schizophrenia or temporal lobe lesions.

*Gustatory hallucinations* are relatively uncommon and usually associated with olfactory ones.

*Tactile hallucinations* are found in toxic states, such as delirium tremens and cocaine addiction. Sexual tactile hallucinations are common in schizophrenia.

*Kinesthetic hallucinations*. Phantom limbs and other body-image disturbances, such as alterations in size, shape or movements of bodily parts, are all included under this caption.

## DISORDERS OF THINKING

The joining or fusion of ideas by various mental processes and the formation of new ideas constitute the function of "thinking". Thought disorders

may be of the nature of disorders of (1) production of thought, (2) progression of thought, or (3) contents of thought

### *Disorders of Production of Thought*

Normally the process of "thinking" takes places at various levels. In *rational* or *realistic* thinking, stimuli are derived from conscious, unconscious and affective sources, and are corrected by reason and logic. Such rational thinking is quite different from *austistic* or *dereistic* thinking, which is found in schizophrenia. Dereistic thinking is dominated by unconscious factors, complexes and drives, and lacks the checking influences of reason, logic and reality. Thought production may be increased or decreased, as can be inferred from increased or decreased pressure of speech.

### *Progression of Thought*

The rate and manner of progression of thought can be assessed or inferred from the "stream of talk." This is normally logical, coherent, uninterrupted without digression, from the initial idea to the goal. Following are the common disorders of progression of thought.

The phenomenon of *flight of ideas* is usually associated with maniacal excitement. This is characterized by increased associative activity, high distractibility and rapid digression from idea to idea, with the result that the person affected is unable to sustain his attention on any "goal idea." Often, words similar in sound to others may conjure up new thoughts. The last word of the previous sentence may become the stimulus to a new idea. This phenomenon is referred to as *chance association* or *clang association*.

*Retardation*, in contrast to "flight of ideas", is characterized by slow initiation and movement of thought, resulting in *slow* speech with a low tone. This is usually found in states of depression and withdrawn schizophrenia.

*Perseveration* is characterized by persistent repetition or continuance in the expression of an idea. The idea is repeated over and over again like an old or damaged gramophone record. This condition is common in organic states such as aphasia, senile dementia, etc. It may also be noted in catatonic schizophrenia.

By *circumstantiality* is meant an inability on the part of the person to reach the "goal idea" without digressing or going round and round in circles. It is due to inability to form clear concepts or discriminate between essentials and nonessentials. It is commonly found in epileptics, mental defectives and in degenerative conditions.

*Incoherence* is characterized by the running off of one idea into another, resulting in disjointed phrases or sentences. This is found in delirium and states of confusion. A lesser degree of incoherence, called "scattered" thinking, is typical of schizophrenia.

*Blocking* is characterized by sudden interruption of progression and expression of thought. The individual momentarily feels that there is a "vacuum" in the brain and struggles hard to pick up the thread. Even normally, a person under stress, with feelings of anger or fear, may experience blocking. Blocking of severe degree is more or less pathognomonic of schizophrenia.

### *Disorders of Thought Content*

Ideas are determined both by logical reasoning and by unconscious and affective factors. When affective factors or "feelings" are predominant, the ideas are described as overvalued or over-determined. When an "idea" or "belief" is totally determined by affective factors and the unconscious defensive needs of the individual, and is devoid of all reason or logic, it is described as a *delusion*. Traditionally, a delusion has been defined as a *false belief*, which cannot be corrected by reason or argument and which is not in tune with the educational and cultural background of the individual. Fairy tales, myths, superstitions, etc. are not delusions, and yet in a small way, they do serve the same purpose of warding off anxiety and affording support or security to the individual. Wishful thinking and prejudice belong to the same category.

The trend and type of delusion are determined by the prepsychotic personality of the individual, which in turn depends on his life experiences, problems and needs. The nature of a delusion frequently betrays some significant aspect or other of the patient's problems. A delusion, which serves the purpose of warding off anxiety or insecurity and of preserving self-esteem, is "defensive" in character. Elaborated ideas, developed from false premises, are known as *systematized delusions*. Delusions are classified as follows: Delusions of (1) Grandeur, (2) Unworthiness, (3) Persecution, (4) Sin, guilt, impoverishment or illness.

*Delusions of grandeur* usually arise from basic feelings of inadequacy, insecurity or inferiority. They are commonly found in mania, G.P.I. and sometimes in schizophrenia.

*Delusions of self accusations or ideas of unworthiness*, which are common in endogenous depression, usually betray a person's sense of guilt about repressed or forbidden ideas and urges.

*Delusions of persecution* are common in paranoid psychoses and in schizophrenia with prepsychotic paranoid personality. They are often associated with ideas of grandeur. In paranoid delusions, the individual is projecting his own aggression, hostility and guilt on the environment. Overambitiousness, coupled with inability to accept defeats and frustrations, resulting in brooding, distrust, suspicion, misinterpretation, resentment or ideas of persecution.

In *ideas of reference*, the patient misinterprets the remarks or actions of others as significant, even when they are not. Feelings of guilt in states of

depression, and projection of one's own self-criticism on to the environment in paranoid states result in ideas of reference

*Passivity feelings* or *ideas of control* are said to exist, when the patient has the delusion that he is being controlled by various magical or ultrascientific devices or that his mind is being read by others. Such feelings are common in paranoid psychosis and depression. *Ideas of sin, disease* or *poverty* are commonly noted in endogenous depressions. The patient may have the delusion that he is a great sinner and should be handed over to the police or even hanged. He may believe that he is suffering from cancer (or some other incurable disease) or that he is a pauper (and has no money even for the next meal) when he happens to be a millionaire. *Nihilistic* ideas are said to exist when the patient exhibits a subjective sense of "absence" or believes that he has no "feeling," "body" or "brain," or that "he is dead"

*Ideas of Suicide* Although transient thoughts of dying or suicide are common, obsessive ruminations about such matters are pathological

Impulses and ideas of suicide are usually associated with ideas of self-depreciation, sinfulness, unworthiness, hopelessness, etc. as in states of depression. Sometimes, they are associated with ideas of grandiosity or yearnings to be united with some person lost through death

Suicidal ideas often represent a wish to fix guilt through revenge, a hostile gesture to some person who has or is fancied to have abandoned, hurt or let down the suicidal person. It may also be an effort to influence the environment and provoke sympathy or attention. Suicidal attempts are common in adults suffering from depression, schizophrenia and personality disorders (like psychopathy and hysteria) and occur at times in delirious states

*Hypochondriac ideas* are characterized by exaggerated concern about one's body or physical health. The patient describes a wide variety of symptoms and believes that he has some serious or incurable illness, even when none can be demonstrated. Hypochondriac ideas are usually associated with depressed affect and found in endogenous depression. They may also be associated with schizophrenia or anxiety states, in which case the prognosis is relatively poor

*Obsessions* Thoughts, that persistently thrust themselves into consciousness against the conscious desire of the patient, are known as obsessions. The person has neither control nor awareness of their source. Obsessive ideas are often related to compulsive behaviour, and are common in *obsessive compulsive neurosis*, but may also occur in states of depression and schizophrenia

*Phobias* are morbid fears, which constantly thrust themselves into consciousness. They are associated with morbid anxiety and may be related to dirt, disease, darkness, crowds, loneliness, lightening, etc. Phobias are fairly common in children and are usually but passing transitory phenomena. Clinically, they are noted in phobic neuroses associated with anxiety states



## DISORDERS OF AFFECT

Affectivity is probably the most important aspect of mental function. It is related to the "feeling life" or "emotional feeling tone" of the individual. Strong, temporary variations, modulations and expressions of emotion or feeling are referred to as *affects*. By *mood* is meant a sustained and constant affective state of considerable duration. This affectivity penetrates and colours the entire psychic life of the individual and influences both cognitive and conative aspects. A mood, to be classed as *normal*, must be adequate, appropriate, reasonably even and neither depressed nor elated. The common disorders of affect are as follows:

*Excitement* Excitement may be mild, moderate or severe, as in euphoria, elation and exaltation.

*Euphoria* is a pleasurable affect, characterized by a feeling of well-being, optimism and confidence. It is found in hypomania, schizophrenia and certain organic diseases, such as GPI, disseminated sclerosis and frontal lobe tumours.

*Elation* is characterized by enjoyment and self-confidence out of proportion to reality and is associated with extra drive or motor activity. It is quite labile and tends to shift to irritability. This condition is common in early manic states, schizophrenia and GPI.

*Exaltation* is an intense excitement, with grandiose ideas and associated with excessive drive and motor activity. It may result in severe exhaustion or even a delirious state. This is found in severe manic states and catatonic excitement.

In *ecstasy*, the mood is one of peaceful rapture or tranquil sense of power. Intense religious feelings and fervent love or affection are common accompaniments. Ecstasy is associated with dissociative, epileptic, schizophrenic and affective reactions.

*Depression* is an affective feeling of sadness, and may range from mild down-heartedness or a mere feeling of indifference to a feeling of intense despair beyond hope. Anxiety, bodily complaints, hypochondriasis, suicidal ideas and even delusions may be associated with this condition, which may be endogenous or reactive.

*Grief* is an affect of sadness, usually suffered through the loss or passing away of a close relative or friend. It is unassociated with feelings of guilt or shame.

*Inadequate affect* By dullness, bluntness, flatness or coldness of affect is meant inadequacy of feeling or affect. In this condition, the person's ability to experience emotions, such as pity, joy and sorrow, are reduced significantly. An apathetic or empty facial expression is associated with lack of drive and interest. Blunting of affect is an early and important sign of the schizophrenic reaction.

*Inappropriate affect* is a common disturbance in schizophrenia. The patient laughs or cries, when actually the opposite emotion is indicated.

*Affective ambivalence* is characterized by contradictory or opposite feelings and attitudes towards the same object. One of the two components remains repressed and is projected later.

*Affective or emotional lability*, also known as "emotional incontinence", is characterized by sudden changes of mood. It is found in organic and degenerative conditions.

*Anxiety* is a persistent feeling of dread, apprehension of impending disaster. It is an experience of *fear*, without any external danger, but due to internal unconscious problems. Anxiety, that is directly felt and expressed in this manner, is known as *free floating* anxiety in contrast to *covert* anxiety. Anxiety is one of the central pillars of modern psychopathology and is the presenting symptom of anxiety neurosis. Through its effect on the autonomic nervous system and endocrine glands, it is capable of disturbing the physiology of all internal organs and systems. In its acute form, it produces hyperventilation, spasms of the cardiac and pyloric portions of the stomach, intestinal irritability, hyperchlorhydria, diarrhoea or constipation, palpitation, tachycardia, extrasystoles, vasomotor flushing and respiratory distress. There may be fainting, weakness, tremor, nausea and sweating of the hands and face. The patient appears unduly tense and displays excessive vigilance and fidgety movements of the hands and feet. His voice may appear uneven or strained and the pupils dilated. When anxiety is severe enough to overflow into the muscular system, it results in severe motor restlessness, known as *agitation*.

*Tension* is characterized by tense emotions and tautness of muscles, associated with restlessness, dissatisfaction, dread and discomforting expectancy. Difficulty of concentration, tightness around the head, tremulousness and strained or tense expression of fear are common accompaniments.

*Panic* is a pronounced or exaggerated form of anxiety, that is productive of disorganization of ego-functions. There is usually a background of long-standing insecurity.

By *depersonalization* is meant a pervasive and distressing feeling of estrangement. This may be of one of two types: (1) a feeling of change in one's own personality, or (2) a feeling that the outside world is not real or has changed. This condition is found in various neurotic and psychotic states.

## DISTURBANCES OF CONSCIOUSNESS

Consciousness can be described as clear-mindedness, when the sensorium is clear. The individual is able to understand his environment as to place, time, persons and general setting as is also able to understand questions and reflect on them. Full consciousness demands attentiveness and perception.

Disturbances of consciousness have different grades of severity and include states of confusion, clouding of consciousness, delirium, fugue states and stupor. *Confusion* is characterized by bewilderment, perplexity, disorientation, disturbances of associative functions and poverty of ideas. It is commonly found in toxic-infective conditions and organic syndromes.

*Clouding of consciousness* is characterized by an impaired capacity to think clearly and rapidly, to perceive, respond to or remember current stimuli. The attention of the patient tends to wander, and understanding of the environment becomes incomplete and inaccurate. Such a state is commonly associated with cerebral anoxia, metabolic disturbances and dissociative reactions.

*Delirium*, also known as "acute brain syndrome" is characterized by clouding of consciousness, bewilderment, restlessness, confusion, disorientation, incoherence, dreamlike thinking, illusions, hallucinations and apprehension or fear. It is commonly associated with organic conditions such as toxic states, metabolic disturbances and cardiac decompensation. It is usually more pronounced during night time.

By *twilight state* is meant a disturbance of consciousness, in which the patient is not aware of his surroundings and tends to have visual or auditory hallucinations. Such a state may last for minutes, hours or days. It may be associated with epilepsy or a dissociative reaction.

*Stupor* is an extreme form of disturbed consciousness, either secondary to some organic or toxic-infective state or to some psychogenic condition such as catatonia or depression. In the organic or toxic-infective form of stupor, conscious thought-processes are usually suspended, while in the depressive and catatonic forms of stupor, such is not the case. A sudden change from stupor to activity (often impulsive or excessive) occurs in the psychogenic form of stupor only, and particularly in the catatonic variety.

*Orientation*—The process by which an individual appreciates his environment and locates himself in relation to it, with reference to time, place and persons, is known as *orientation*. Orientation is usually disturbed in acute cerebral insufficiency and in delirious states, when the physiological functions necessary for memory, perception and attention are impaired. Orientation may also be disturbed through acute conflicts, intense emotions and lack of interest or attention.

*Memory*—The function, by which data acquired and presented to consciousness through observation or attention are stored, and later summoned and presented again to consciousness, is referred to as *memory*. Memory consists of three processes or stages: (1) Registration, (2) Retention, and (3) Recall. It is adjustive and adaptive, by virtue of its role of assisting the individual to profit from experience. Memory is strongly influenced by affect. The three main disorders of memory are

- (1) Hypermnnesia or abnormally sharp memory
- (2) Amnesia or loss of memory
- (3) Paramnesia or falsification of memory

**Hypermnnesia** is encountered in mania, paranoia and catatonia

**Amnesia** may be organic or psychogenic. In psychogenic amnesia, recall of stored data is inhibited unconsciously in order to avoid some unpleasant memory (e.g. an unbearable or shameful experience), promise or appointment. It is as a rule selective, usually restricted to circumscribed periods and events, and has a tendency to recover suddenly and completely. Psychogenic amnesia is associated with various hysterical manifestations. In amnesia of *organic* origin, the functions of registration and retention are disturbed. Loss of memory for recent events is characteristic of this type of amnesia. A *fragmentary* type of amnesia occurs in G.P.I. and senile dementia. There may be a *generalized* loss of memory, for both recent and remote events, in cases of degenerative disease of the brain. Lastly, amnesia may occur in association with confusional states, epileptic episodes, delirium and head injuries. Recovery from organic amnesia, when it does occur, is usually gradual and incomplete.

*Anterograde amnesia*, which extends forwards in time and experience, beyond the initial amnesic event, is found in boxers and in Korsakoff's psychosis.

In *retrograde amnesia*, there is loss of memory extending backwards, over a period prior to the time of onset of the attack. Retrograde amnesia occurs after head injuries, hanging, gas-poisoning, and after electric convulsion therapy (E.C.T.). Occasionally, it is psychogenic in origin.

**Paramnesia** is characterized by falsification or distortion of memory. Confabulations or stories are made up, in which the patient fills in the gaps with fabrications (which change from moment to moment). This condition, although typical of Korsakoff's psychosis, may also occur in senile psychosis.

*Retrospective falsification* is characterised by misinterpretation of past events, through remembering certain details and forgetting others, according to one's own emotional needs.

The *Deja Vu* phenomenon is an illusion of the memory, in which there is a vague feeling of familiarity, as if one has undergone a similar experience in the past, although in fact one has not. This is common in both normal and schizoid individuals. In the *Jamais Vu* phenomenon, on the other hand there is a false feeling of unfamiliarity with situations, which have actually been experienced. This may be observed in schizophrenia, neuroses, toxic states, fugue states and temporal lobe lesions.

**Dementia** is characterized by a permanent and irreversible loss of intellectual functions. Early dementia of mild degree is characterized by disturbed critical faculty, lack of power of discrimination, abstract ideation, etc. As

dementia progresses, there are added poverty of initiative, restriction of interests, blunting of concern, and progressive deterioration and loss of other intellectual functions (such as learning, memory, grasp, orientation and judgement) Emotions become labile and inadequate Dementia is caused by structural disturbance or degeneration of higher cortical neurons, secondary to prolonged intoxication, malnutrition or degeneration It may also be associated with traumatic, infective and neoplastic conditions of the brain.

## PSYCHIATRIC HISTORY

The purpose of history-taking in psychiatry, as in the rest of medicine, is primarily *diagnostic* The important role of history-taking in facilitating diagnosis is seldom realized. In psychological medicine, the history is even more important than in physical medicine Not only is it important in determining the nature of symptoms and grouping them into syndromes in order to arrive at a clinical *diagnosis*, but also in evaluating the sources of conflicts and stress and their interaction with the individual's personality in order to determine the *pathogenesis* of the condition. In order to understand mental "stresses" and evolution of personality traits, one has to study not only the patient's *present* life, but also his entire *past* life, from the time of conception to the onset of illness Such a biographical understanding of the individual enables the clinician to have an insight into the dynamic evolution of the personality, its deviations from normal and the nature of the illness The psychiatrist must not only be a clinician, but also a psychopathologist A dynamic diagnosis (rather than a mere clinical "label") frequently suggests not only the outlook or prognosis of the case but also the right therapy In this way, history-taking is of utmost importance in psychiatry, and every student must try to master the correct technique A diagnostic interview not only determines the type of future therapy indicated, but may actually prove of therapeutic value A psychiatric patient frequently feels better and more comfortable after the first interview A diagnostic interview must be conducted with great care, the important issues involved being (1) The doctor-patient relationship, and (2) The mode of interrogation

Information obtained (about the patient's illness, change of behaviour or pre-illness personality) from the patient himself, is as a rule not dependable in psychiatric history-making, because of the likelihood of disturbed mental function or lack of insight into one's own illness Any information received from the patient himself must therefore be confirmed or supported by some near relative or friend, who is reasonably objective in his thinking and genuinely interested in the welfare of the patient No psychiatric history can be considered adequate or reliable unless this is done A "positive" doctor-patient relationship is important in psychiatric work (as in the rest of medicine), although it is often neglected to the detriment of the patient's interests Understanding and co-operation from the patient, which are so vital for the success

of any therapy, can be achieved only through a positive relationship. The doctor-patient relationship depends both, on the doctor's attitude to the patient and his illness, as well as on the patient's attitude to the illness and towards his doctor.

A psychiatrist's attitude to mental illnesses and patients frequently depends on his own personality, as well as on his knowledge and ideas about psychiatric ailments. Some doctors are peculiarly negligent and indifferent towards mental patients, an attitude that usually springs from prejudice or ignorance. This attitude has done considerable harm to many a psychiatric patient with so-called "functional" illnesses. These are *real* illnesses, involving as much suffering, discomfort and pain as organic illnesses, and the psychogenic bodily symptoms therefore deserves the same sympathy and attention that is afforded to other patients. Psychiatric patients are not aware of the causes of their ailments, nor they are responsible for them, like patients with organic illnesses. The student must bear these facts in mind and cultivate a scientific, rational and human attitude to such illnesses and patients. The examiner must learn to overcome the barriers of language, culture, status and social class, commonly encountered in public hospitals due to intellectual, social and cultural differences between the doctor and patient. For a positive relationship, the examiner must inspire confidence in the patient, put him at his ease and make him feel that there is a friendly expert who is genuinely interested in his welfare. He should also be willing to accept and tolerate certain indifferent, non-co-operative, negative or hostile attitudes, arising out of such illnesses. By avoiding counter hostility and aggressiveness, he not only helps the patient but saves his own energy and avoids discomfort and inefficiency. In view of the stigma or shame frequently associated with mental illnesses, the patient's attitude towards his illness is one of secretiveness or denial. This leads to marked secondary anxiety, depression and a feeling of frustration, frequently resulting in non-co-operative and abnormal behaviour towards the medical fraternity. It is the doctor's function to help the patient to overcome his disability by correct orientation, education and handling of the patient. Relatives of psychiatric patients are sometimes difficult and non-co-operative, being dominated by feelings of shame, denial or hostility towards the patients. Their personalities, motives and attitudes to the patients must be assessed, before their versions are taken as objective and true. Often a relative's attitude to the patient and his illness is totally negative and full of hostility for various reasons, which need understanding and help. The examiner must therefore learn to overcome certain difficulties about himself, the patient and his relatives, in order to create a proper atmosphere and a positive doctor-patient relationship, conducive to successful diagnosis and therapy.

A *diagnostic* interview must be so conducted, that it yields the maximum amount of information and serves to establish a firm and positive relationship or rapport between the physician and patient. There can be no rigid rules

or "pattern" of interview applicable to all patients. It must vary with the doctor and the type of the patient. Sympathy, apathy, objectivity, flexibility and sincerity are some of the many attributes necessary for success.

Absolute privacy is very essential. A psychiatric history, taken in open out-patient clinics, with people moving around, has hardly any value or meaning. Since private, personal, delicate or emotionally charged matter has to be verbalized, an atmosphere conducive to such expression is necessary. Patience, interest and attention, on the part of the doctor, are necessary. It is always best to let the patient have his own say first, and then, whatever information that appears lacking is collected through conversational channels rather than through formal questions and answers. In the beginning, it may be necessary for a student to write down the details of the history during the interview, but ultimately he must learn to master the technique of documenting a history, immediately after the interview with the patient. A history that is recorded after a lapse of days or weeks is likely to be distorted and unreliable.

For the documentation of a history, a standardized scheme or proforma is necessary, but during actual history-taking each patient is examined or questioned individually, on his own merits. Feelings of guilt, shame or fear, involved in describing one's private and personal emotional life, may prevent some individuals from being frank or open in their interviews. This resistance or negative attitude must be handled quietly and overcome by gradually establishing positive relationship and without resort to impatience, arguments, loss of temper or hostility. For this reason, it is necessary to start the interview with only those points in which the patient is primarily interested, and then explore areas in which the doctor is interested. Even here, one should start with peripheral, general and less personal matters, and ultimately come to central and highly confidential, emotional topics.

## CHIEF COMPLAINTS WITH ORIGIN, DURATION AND PROGRESS

A psychiatric history, like any other medical history, should start with a presentation of the "chief complaint" or "problem", by the patient and his relatives, in their own words. The doctor may at times have to guide the patient and relatives along the right channels or prevent them from dwelling too long on irrelevant or useless data. In principle, it is always best to allow the patient to describe his own problem freely in his own words, and to his satisfaction, regarding the origin, duration and progress of the illness. This is then followed by active questioning by the doctor, with a view to elicit other relevant points or missing information and fill up the "gaps" in order to facilitate diagnosis. It is important to inquire about the treatments carried out up-to-date.

While discussing the present illness one must inquire about the previous health and past illnesses of the patient. A history of head injuries, meningitis, encephalitis and syphilis, as well as of other psychosomatic illnesses, must be inquired into.

#### FAMILY HISTORY

After obtaining a history of present and past illnesses, it is desirable to concentrate on the "family background" of the individual. The financial, educational, cultural and social status of the family, the number of family members and their personalities, and their relationships with each other and the patient must be ascertained. Special inquiry must be made about the mental and physical health levels of the family members. Care must be taken to ascertain the presence or absence of certain diseases, such as mental defects, schizophrenia, manic depressive psychosis, epilepsy and hysteria in family members.

#### PERSONAL HISTORY

The family history is followed by a detailed personal history, with a view to ascertain the development and nature of the pre-illness personality. The sources and types of stresses and conflicts, the way these are handled by the patient and with what personal and interpersonal consequences, must all be enquired into carefully and documented, as these may help to formulate the psychopathogenesis and dynamic diagnosis of the illness. Documentation of the personal history may be done in a chronological order, involving various aspects of living like infancy and childhood (from parents or other senior relatives), followed by school and college history, occupational and financial history, social history and psychosexual history. Questioning of the patient however may be varied according to the type of illness and the patient. It is best to start with present, factual data, such as occupation, interests and education, and then work up slowly to more delicate or private areas, such as psychosexual development and sexual life.

**Infancy and childhood** Information must be obtained about the parents' needs and attitude towards conception. The birth history, whether full term or not, whether normal or abnormal and whether associated or not with injury, etc must be recorded. Physical illnesses during infancy or childhood, such as head injuries, convulsions, neonatal jaundice, and encephalitis, must be inquired into. Further enquiries must be made about general health and personality attributes of the patient, his relationship with family members and playmates, the amount and types of games or play indulged in and presence of psychosomatic symptoms and neurotic traits. Further details about breast feeding, weaning, type of toilet training, general disciplinary measures and separation from parents must be noted.

**School and college.** The patient's attitude towards schooling, his scholastic performance, relationship with teachers and other pupils, participation in



extra-curricular activities and attitude to pupils of the opposite sex, reasons for stoppage of education and attitude to it should be inquired into

**Occupational and financial history** It is important to know whether the patient's present *occupation* is of his own choice or thrust upon him by others. His attitude to the job, whether or not he enjoys it, his efficiency and performance, his relationship with seniors, juniors and colleagues, whether he gets his regular promotions or not, how many jobs he has already changed (and if so, why?), all are questions worthwhile pursuing. A frequent change of jobs, without progress or promotion, suggests that the patient is probably an unstable and drifting type of individual, probably ailing with psychiatric illness.

For a woman, managing the house, children and other domestic and social affairs is just as important and difficult an occupation as a man's job in the office, factory or shop. It involves the same amount of stress and difficulties as any other manly job. The amount and quality of domestic work, upbringing of children, relationship and conflicts with domestic servants (if any) must all be enquired into.

Financial history is very important in a country like ours, where financial insecurity and stress are so common and play an important role in causing psychiatric disorders. The income per capita, expenditure, debts, and the attitude of the patient towards these items, are all to be ascertained.

**Social and recreational history** Whether the patient, before his illness, was a good mixer? Had any friends, hobbies or general interests? Pattern of spending the leisure hours and nature of recreations. Exercise, sports, interests in cultural activities, such as dramatics, music, dancing, painting, movies, etc.? Other interests, like politics or religion? Whether the patient's personality is broad-based, with wide interests and contacts or the other way about?

**Psychosexual history** Sex has been traditionally subjected to more control regulation than any other instinctual need of the individual. It is therefore not surprising that a large number of people have conflicts and problems relating to sex. These are further aggravated by the privacy, secretiveness, taboos, prohibitions, ignorance and superstitions widely prevalent about sex matters. Being a delicate subject, the approach to sex problems must be gentle and inoffensive, starting from socially approved and legitimate activities such as nocturnal emissions, masturbation, homosexual experiences, etc. to the socially disapproved ones. Later, enquiries are made about other activities, which may be fear, shame or guilt-provoking. The attitudes and conflicts about such activities must be noted. The majority of patients have ample opportunities to unburden themselves regarding various problems of life, but seldom have the chance or opportunity to discuss or verbalize difficulties in the sexual sphere. Hence, most patients with sex problems on their

minds, are keenly awaiting or looking forward to an opportunity (such as a psychiatric interview) for seeking medical advice on their problems. The doctor must therefore take the initiative and inquire into details about this aspect of life.

**Marital history.** Whether married or unmarried? If married, for how many years? How many children? Marital relationship. First meeting, engagement, courting period, honeymoon and reaction to it. Sexual life—pattern and attitude. Attitude to pregnancy and family planning. Sources of disagreement, conflicts or stress in marriage. Personality of husband or wife.

After the marital history, one may inquire, in the case of *men*, about the onset and frequency or nocturnal emissions and their attitude towards them, sex education (when and how much), attitude to sex in general, practice of and attitude to masturbation, and whether accompanied by feelings of shame, guilt or fear. Homosexual experiences if any. Love affairs, premarital and extra-marital affairs if any and attitude towards them.

In *women*, sex education, onset of menstruation, preparedness and attitude towards menstruation, attitude to femininity. Indulgence and attitude to masturbation. Love affairs, premarital and extra-marital relationships (if any) and their attitude towards such digressions.

**Personality traits and type.** After this, an inquiry is made into the various traits of the patient's personality with the help of near relatives and friends. This serves to construct the pre-illness personality.

The purpose of amassing so much data about the patient's personal life is to make one understand the pathogenesis and meaning of symptoms. At the end of the history-taking, the examiner should be able to formulate the type of personality of the patient, its evolution from birth to the onset of illness, the stresses and conflicts it has had to contend with, and the way in which these have been faced or handled, ultimately to result into symptoms.

## PSYCHIATRIC EXAMINATION

Psychiatric examination starts from the moment the patient enters the room for the interview and is being continuously carried out by the psychiatrist during the entire history-taking. Documentation of data is carried out as follows.

**Attitude and manners.** The way the patient enters, moves, walks, stands, talks and behaves. His attitude to the doctor and illness. Co-operative or non-co-operative? Patient's dress, hair, style, cosmetics and personal cleanliness. These items are judged in terms of his pre-illness personality as well as the accepted standards of the group to which he belongs. Observation and study of external appearances are of great value in diagnosis, particularly of psychoses (like mania), depressions and schizophrenia.

**Consciousness** The state of consciousness, whether clear or cloudy and to what extent, it is deranged or lost

**Affect and mood** The type, intensity, depth and duration of the prevailing affect can be judged from the facial expression, muscular tension, bodily attitude, manner and content of talk of the patient. The patient may be fully aware of his disability, as in cases of early schizophrenia, where he may actually complain of a blunting of affect. Patients with hypomania or depression usually have some insight into the affective change that has come over them

**Thought** The thought-processes must be studied, from the points of view of production, progression and content. The pressure of speech must be noted as well as the presence of any irrelevancy, incoherence, blocking, circumstantiality, neologisms, perseveration or echolalia

The patient may himself volunteer information about phobias and obsessions. Otherwise they have to be inquired into. Over-determined ideas and delusions are important disturbances of content and must be evaluated. It may not be easy at times to determine whether a belief should or should not be classed as a delusion. The patient, while describing his complaints, may either reveal the presence of delusions or succeed in hiding them. It requires considerable tact, patience and experience to unearth certain delusions. It is important to determine whether a delusion is transitory, fixed or systematized

**Perception** The main disturbances of perception are illusions and hallucinations. Although the patient may give a good account of such disturbances on his own, one must always try to ascertain whether these are of the nature of illusions or hallucinations. A positive enquiry about hallucinations must be made in a very tactful manner. Even when denied, their existence may be inferred from the patient's behaviour, listening attitude or response to such a hallucination. An account of the patient's behaviour afforded by the nurse or relative may prove useful in deciding the issue

**Memory** As the patient recites the history in chronological order, with dates, names and events, his memory for remote events is usually revealed. Recent memory can be tested by asking him the time of admission to hospital or the name of the person who accompanied him to hospital. Any evidence of confabulation must be noted. Retention and recall are tested by reciting a series of non-consecutive numbers and then asking the patient to repeat them in the same or reverse order. The results of such a test are not reliable, if the patient fails to co-operate fully with the examiner. An objective history about the patient's memory from a near relative is often quite useful in this regard

**Orientation** Orientation must be tested in terms of time, place and people. This may be tested by asking the patient the day of the week, the date or the

month, and by enquiring whether he manages to find his way to the office or home and whether he recognizes all friends and relations properly

**Intelligence.** Intelligence can be judged clinically during the interview, by the way the patient presents his problems and personal history. The logic and reasoning, he displays during the interview and the fund of information at his command are fair indications of his intelligence. Questions relating to current events, popular matters and political news, may be asked in order to assess his fund of information or knowledge. An easy way of determining the individual's intellectual level is to make enquiries about his school performance for several years. A consistently high or low rank in class usually indicates a high or low level of intelligence, provided the school is reliable and determines the rank in class on the basis of merit alone. An emotionally disturbed child or even a gifted child with high intelligence level may at times be at the bottom of the class. Determination of the level of intelligence is particularly important in intellectually handicapped individuals.

**Judgment.** By judgment is meant the ability to compare facts or ideas, to understand their relationships and to draw correct conclusions from them. This can be tested or inferred by inquiring about the patient's future programme and whether he is able to conduct his business affairs with prudence and meet his family obligations.

**Insight.** Insight refers to the individual's knowledge of self, or his ability to observe and understand himself. Awareness of one's own illness and of its nature and dynamics constitutes *insight*. Insight being usually absent in the psychoses, patients with these disorders are often non-co-operative and tend to refuse all treatment. It is *insight* that makes a patient co-operative and willing to receive treatment, thus improving the prognosis or outlook of the condition, particularly under psychotherapy.

The clinical history and examination, for practical reasons, are best finished in one session. At times, it proves necessary to explore the patient's life for several sessions, before one gets sufficient data for a comprehensive, clinical, genetic and dynamic diagnosis. In case, the patient is unable to divulge or reveal personal features, because of aphonia, amnesia or non-co-operation, resort may be had to special techniques, such as examination under narcosis or hypnosis. By employing disinhibition or suggestions in this manner, the patient may be made to give more pertinent data about his disease. Even when inaccessible, unwilling or unable to communicate linguistically, a patient may unknowingly convey a lot of information about his mental state through positive or negative behaviour, which the student must learn to note carefully during the interview. The general reactions, movements, posture, facial expressions and emotional responsiveness of the patient must be observed and recorded. A diagnostic formulation is only possible after a thorough history and examination of the patient.

The diagnosis of a psychiatric case should be in terms of clinical, genetic and dynamic diagnosis. Assignment of an exact position to the disease-process in the traditional classification of diseases, on the basis of the symptom-complex displayed by the patient, constitutes *clinical diagnosis*. The *genetic* diagnosis takes into account the vulnerability of individual to genetic or hereditary, organic and childhood handicaps. The *dynamic* diagnosis deals with pathogenesis and the conflicts and stresses responsible for the disease-process.

# 13 | The Nervous System

**Introduction** The diagnosis of any disease process involving the central nervous system implies a determination of the *site* of the lesion as well as its *nature*. A complete neurological examination, in a systematic manner, is therefore essential in any case suspected of having a pathological lesion of the nervous system.

In the case of neurological disorders, it is as a rule easier to localize a pathological lesion and determine the manner in which it affects function, than in the case of the other systems of the body. The value of a neurological examination depends on a careful interpretation of the patient's symptoms, a proper elicitation of physical signs and on the correlation of observed data with a knowledge of the anatomy and physiology of the central nervous system. The mode of development and natural history of a disease process may shed valuable light on the nature of the disease.

In every case, a neurological examination must always be supplemented by a thorough examination of other systems of the body.

## HIGHER FUNCTIONS

**Introduction** An evaluation of the mental or intellectual state of the patient is important, at the beginning of every neurological examination, as it may shed valuable light on subsequent findings. For instance, a patient with a defective memory cannot be relied upon to give a dependable history of his ailment, similarly, in the case of a drowsy, delirious or semi-comatose patient, or one who has lost his powers of speech, or is unable to understand the language of the examiner, an examination of the sensory system may be unreliable or impossible.

**Method of investigation** A number of highly elaborate tests have been described for investigation of the higher functions. The student is however advised to depend on simple methods of examination (described below), and correlate his findings with the information obtained from the patient's relatives, and from his own observation of the patient.

It is important to realize that disturbance of the mental state of a patient may result, quite apart from organic brain disease, from excitement nervousness, depression or psychosis

Although it is customary to carry out tests of higher functions at the beginning of a neurological examination (for reasons mentioned above), there are some who prefer to carry them out at the end of the examination, according to them, most defects involving higher functions manifest themselves clearly and do not require special methods of investigation, during routine physical examination of the nervous system

Right or left-handedness Clinical experience has shown that with respect to language function, one hemisphere is invariably dominant to the other. In the majority of right handed individuals, the left hemisphere is dominant. In the left handed individuals the dominance is less clear cut and in many, the left hemisphere may be dominant. Dominance is probably genetically determined. Since most children are compulsorily instructed to use the right hand in writing it is no use asking them which hand is used for writing. It is best to ask which hand is customarily used by the patient to comb the hair, thread a needle, throw a ball or pull the cork out of a bottle. The importance of this is that in case of loss or abnormality of speech function, the knowledge of cerebral dominance in that individual helps in localising the lesion to the appropriate hemisphere.

Examination of the mental state An evaluation of the mental function is an integral part of every complete neurological examination. The mental functions of a person are commonly disturbed by neurological disorders. Such a disturbance may result directly from damage to the brain, indirectly from the emotional strain imposed by the illness or from a combination of both these factors. The headings and methods described below should be followed for the sake of uniformity but the data may be collected initially with inquiry about the present illness of the patient. The state of the patient with regard to the following should be ascertained.

### General behaviour

Consciousness and interest in surroundings Responsiveness of the patient to painful, tactile, thermal and visual stimuli presented to him should be observed. The level of consciousness for purposes of description can be divided into four grades

(a) Somnolence The somnolent patient may be roused by various stimuli and will then make appropriate motor and verbal responses. When aroused such a patient may be clear mentally but often is somewhat confused.

(b) Stupor The stuporose patient can be aroused by painful stimuli and after repeated stimuli may respond for short periods to simple commands. Restlessness and spontaneous movements are common.

E ENJOYMENT  
I - Intelligence  
C - Continuity  
U - Understanding / Delusion

(c) Semicoma In this state painful stimuli or shaking will cause withdrawal or other adaptive movements Muttering is not uncommon As soon as the stimulation ceases the patient reverts to his original state

(d) Coma The patient is deeply unconscious and there is no response to any stimulus or there is only slight response to a very painful stimulus such as pressure over the styloid, supraorbital nerve or root of the finger-nails

Gestures, grimaces, mannerisms or other motor expressions should be observed Are they spontaneous or how provoked and whether constant, abrupt or fitful?

Attention Is attention easily obtained, kept or can it not be aroused, or is it apt to wander? If inactive, does the patient resist passive movements, or obey commands, or indicate awareness at all?

Eating habits, sleep, cleanliness in general should be found out

Talk The form of the patient's utterances rather than their content is considered Does he say much or little, talk spontaneously or only in answer, slow or fast, hesitantly or promptly, to the point or wide of it, coherently, loosely or with interruptions? Note if any sudden silences A sample of talk should be recorded It should be representative of the form of his talk, his response to questioning and his main preoccupations

Mood The patient's appearance may be described so far as it is indicative of his mood His answers to "How do you feel in yourself?" How about your spirits or some similar inquiry should be recorded. Many varieties of mood may be present, not merely happiness or sadness, but such states as irritability, suspicion, fear, unreality, worry, restlessness, bewilderment and many more Observe the continuity of the mood, the influences which change it, the appropriateness of the patient's apparent emotional state to what he says

Delusions and Misinterpretations What is the patient's attitude to the various people and things in his environment? Does he misinterpret what happens, give it special or false meaning or is he doubtful about it? Does he think anyone pays special attention to him, treats him in a special way, persecutes him, or influences him bodily or mentally? Does he depreciate himself in any regard, his morals, possessions, health? Has he grandiose beliefs?

If the patient has any hallucinations, are they well-organised images or ill-formed? These matters may be complicated and need much inquiry

Delusions are <sup>false</sup> fake beliefs which cannot be changed by reasoning with the patient or contrary evidence, and which cannot be explained by the social or educational background of the patient

Hallucinations are false impressions arising from the organs of special sense, for which no cause is found Temporal lobe lesions produce well-formed hallucinations, while the visual hallucinations in occipital lobe lesions are ill-formed



Illusions are optical phenomena, which can be scientifically explained, in which the image is distorted or different from the object.

*Compulsive phenomena* Are obsessional thoughts, impulses or acts felt, and if so from without or as part of the patient's own mind? Does their insistence distress him? Does he recognize their inappropriateness? What is the relation to his emotional state? Does he repeat any acts such as washing unnecessarily to reassure himself?

*Orientation* Record the patient's answers to questions about his name and identity, the place where he is, the time of day and the date

*Memory*

(a) *For recent events* such as those of his admission to hospital Any selective impairment of memory for special events should be noted.

(b) *Digit retention.* Give the patient digits to repeat and record how many he can repeat immediately after being told them, e.g.

582, 6439, 42731, 619472, 591728, 58192647

Forward

Backward

The ability to retain seven digits in three consecutive attempts constitutes a satisfactory response

(c) *Reproducing a story* A short story is read over to the patient who is then asked to reproduce the story in his own words and to explain its meaning, e.g. Gilded boy story

'At the coronation of one of the Popes, about 300 years ago a little boy was chosen to play the part of an angel, in order that his appearance might be as magnificent as possible he was covered from head to foot with a coating of gold foil. The little boy fell ill, and although everything possible was done for his recovery, except the removal of the fatal golden covering, he died within a few hours.'

(d) *General information* Question relating to general information may be asked such as the name of the President of India, the Prime Minister, the date of the Independence day, the capital city of the country, the colours of the traffic lights and what they are meant for

(e) *The 100—7 test* The patient is asked to subtract 7 from 100 and to continue subtracting 7 from the result. The observer will make certain that the test is understood and may according to his own judgement give any form of encouragement short of giving the patient the actual number. The time required should also be noted.

(f) *Memory for past events* Is tested by asking the patient about the place and date of his birth, about his school and surroundings in childhood, past hobbies and vocations

*Insight and judgement* What is the attitude of the patient to his present state? Does he regard it as an illness and needing treatment? Is he aware of

mistakes made spontaneously, or in response to tests? Does he recognize and acknowledge any special incapacity he may have? What is his attitude to recovery? What is his attitude towards social, financial, domestic and ethical problems? Is his judgement good? What does he propose to do when he leaves hospital?

**Ability for calculations** The patient's ability to carry out additions, subtractions, multiplications and divisions should also be tested *Acalculia* or loss of ability for calculations may be encountered in organic dementia or parietal lobe syndrome of the dominant hemisphere

## S P E E C H

**Definition.** Speech, as opposed to articulation, is a relatively higher function of the brain By speech is meant the faculty of communicating, expressing or understanding thoughts and ideas through the medium of symbols, which may take the form of spoken or written words Whilst a disorder of the central or cerebral mechanism of speech is referred to as *aphasia* or *dysphasia*, a disorder of the peripheral mechanism of speech, involving articulation or enunciation, is referred to as *dysarthria* Whilst in aphasia the process of understanding or expressing meanings or thoughts is deranged in part or in its entirety, in *dysarthria* or *anarthria* there is a partial or complete inability to convert formulated words into sounds

The term speech implies an expression of one's thoughts and ideas by means of visual or auditory symbols for the convenience of others as well as the comprehension of the thoughts and ideas of others through written or spoken word Speech therefore consists of (1) a *productive mechanism*, dealing with the expression of one's own thoughts and ideas to others, and (2) a *receptive mechanism* which deals with the understanding or comprehension of the ideas and thoughts of others

**Evolution.** It is important to trace the development or evolution of speech and language through the centuries, as well as its development in a child through its formative years, in order to understand and diagnose the various disorders of speech Primitive or prehistoric man, somewhat like an animal, was perforce obliged to employ sounds for conveying his thoughts to others, his speech being limited to the expression of various emotions through the agency of certain *meaningful sounds* With further development of speech, certain sounds became symbolic of or associated with certain objects or people, such as food, mother, dog, etc thus initiating the use of words for the first time, with an increasing vocabulary of meaningful sounds, words were linked into sentences, and sentences into a language *Spoken speech* came first both for expression and understanding *Written speech* must have developed at a much later date, when written symbols were evolved with a view to conveying the meanings of spoken words

The development of speech, in a growing child, follows a pattern closely similar to that of its development in humans through the ages. Emotional sounds with meanings come first, followed by single words of a simple kind, then more difficult words and short sentences, and finally increasing facility of speech throughout life, depending of course on the individual's learning and natural tendency to developing words and sentences. In a child, the understanding of the spoken speech of others usually precedes the formulation of words or meaningful sounds on the part of the child itself. The expression and understanding of written symbols and words follows much later.

**Localization** (Fig 13 1) Speech, being a so-called "higher function", has its centre of location within the brain. Speech is essentially a function of the dominant cerebral hemisphere (the left cerebral hemisphere in right-handed persons). The exact site and mode of action of the speech centre, however, remain controversial. There are some who believe in sharply localized areas, within the brain each area being in charge of a specialized speech function, there are others who deny the existence of any such localization of speech areas in the brain. A rigid localization of speech centres within the brain substance has been firmly believed in until recently, this was because of the fact that disease or damage to certain parts of the brain usually result in certain set patterns of symptoms or disorders of speech. As Hughlings Jackson showed years ago, the localization of a disorder of speech does not necessarily justify a localization of the function of speech. The term "area" of speech being more generic and less specific in localization is preferable to the more commonly used term "centre" of speech.

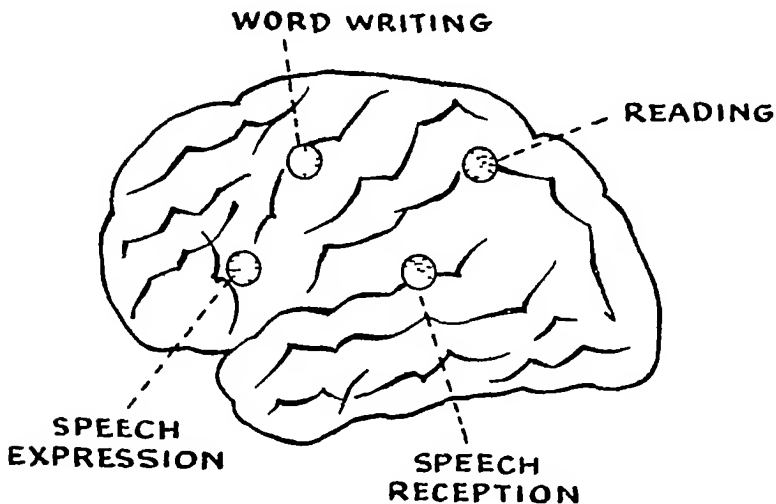


Fig 13 1, The cortical speech areas

On the basis of various studies it is generally accepted that in the majority of right handed individuals the left hemisphere is associated with verbal skills and the right with non-verbal ones. With left handed individuals, very often the left hemisphere is the "dominant" one in this regard and not the right as one might expect.

In right-handed persons, the posterior part of the left superior temporo-sphenoidal convolution, near the auditory centre, is said to be concerned with the understanding of spoken speech, any injury to this centre (auditory speech area) causing auditory sensory aphasia, word deafness or difficulty in the comprehension of spoken speech. The limits of this area have not as yet been clearly defined. It probably includes the inferior and posterior parts of the parietal lobe and adjoining parts of the temporal lobe (Wernicke's area). The pulvinar is closely related to these areas.

The motor speech area, which represents the productive mechanism for spoken speech and which elaborates and coordinates the movements of the mouth, tongue, lips and larynx in order to produce meaningful words, is said to be located in Broca's convolution, in the lower and posterior part of the third frontal convolution.

The visual speech centre which represents the reception or understanding of written speech, is said to be in the posterior part of the first temporal convolution, extending backwards up to and including the angular gyrus.

A special area or centre for writing representing the productive mechanism for written speech, is apparently located in the posterior part of the second frontal convolution, anterior to the cortical area for movements of the hand and fingers.

## APHASIA (DYSPHASIA)

By aphasia or dysphasia is meant a loss or impairment of the cerebral function of speech, or the capacity to use or understand words as symbols of ideas or thoughts. Usually due to an organic lesion of the cerebral cortex, aphasia represents an impairment or loss of one of the highest functions of the brain. Depending on the nature and degree of the speech defect, many different sub-divisions of aphasia are possible.

Disorders of speech (aphasias) are of two main types. When a lesion is posteriorly placed anatomically, the speech defect involves the receptive or sensory mechanism of speech (sensory aphasia), there being defective understanding or comprehension either of spoken speech (auditory aphasia or word deafness), or of written speech (visual aphasia or word blindness). Similarly when the lesion involves the productive or motor mechanism of speech (motor aphasia), being situated anteriorly, there is difficulty in expressing or producing either spoken speech or written speech (agraphia or inability to write). In the majority of cases, disorders of speech are combinations of two or more of these defects.

Sensory (receptive or afferent) aphasia It is the inability to understand the spoken or written words of others. It would be true to say that a man, hearing a foreign language of which he has no knowledge or understanding, has a receptive aphasia of *physiological* rather than pathological type. A patient with organic or *pathological* receptive aphasia, on the other hand, is unable to understand or read a language with which he has been familiar all his life. The receptive defect may be for both spoken and written words, in which case the patient is unable to carry out simple commands, like "open your mouth" or "put your arms up" etc., not having comprehended the meaning of the words. Similarly, whilst reading a newspaper, or even a book, with which he has been familiar all his life, he finds great difficulty in understanding the meaning of words, as a result, he is either unable to read at all, or when reading aloud, sounds incoherent, uttering words and sounds incomprehensible to the examiner. In severe sensory aphasia, with complete lack of understanding of speech, the patient may bring forth, a meaningless jargon (*jargon aphasia*) or neologisms, the lesion being in the temporoparietal area. Lack of understanding of spoken speech is usually a part of very severe receptive or expressive aphasia. When a defect is confined to spoken speech only, the sensory aphasia is referred to as *pure word deafness* or auditory aphasia, in such a case, reading, writing and talking are normal, and the condition is suggestive of a lesion in the posterior part of the superior temporal convolution. When a speech loss is selective for written words, it is referred to as *pure word blindness*, *visual aphasia* or *alexia*, and is usually secondary to a lesion of the parieto-occipital area. In view of the diffuse or wide extent of the sensory speech area (unlike the motor area), even massive lesions may affect the reception of speech selectively. *Developmental alexia* refers to an inability in learning to read.

Expressive (motor or verbal) aphasia This may be for spoken or written words, there being an inability to express one's thoughts to others, through talking or writing. In *pure motor aphasia*, the patient is unable to find correct words and put them into correct grammatical order. His internal speech is usually normal, the patient knows what to say, but when he makes the effort to say it aloud, wrong words come out and he is not able to convey any meaning. Being aware of his disability, the patient tends to become reticent, depressed or even "wordless", uttering only monosyllabic sounds, like "yes" and "no", to the examiner's queries, he may even utter wrong words, saying "yes" when he actually means "no". The lesion, in such cases, is usually situated in the lower part of the precentral convolution and the posterior part of the third frontal convolution (Broca's area).

In *agraphia*, there is a selective loss of writing ability, the patient being unable to write either spontaneously, to dictation or to copy writing, even though capable of conducting a conversation. Several varieties of agraphia have been described and ascribed to lesions of the left angular gyrus or second frontal convolution (posterior part).

AMNESTIC OR NOMINAL APHASIA is a selective form of motor aphasia, in which the patient is unable to recall and utter the right names of objects or persons, unless reminded of them through devious channels. He may describe correctly the nature or purpose of an object, for instance, he may describe a fountain pen as an object with which to write, but cannot utter the words "fountain pen". Again, when several alternatives are offered to the patient by the examiner, as likely names for the object in question, he can usually pick out the correct one for instance, if shown a fountain pen and asked "Is it a glass?", "No", "Is it a knife?", "No", "Is it a fountain pen?", "Yes, yes—that's it, it is a fountain pen". And in that way, he may be able to say the correct words, "fountain pen", which he could not otherwise do spontaneously. In mild cases, the patient may be able to name familiar objects but is unable to name the unfamiliar or less familiar ones. Although a mild degree of nominal aphasia may be observed in the absence of neurological disease, as in nervousness, toxæmia, exhaustion or old age, a pronounced degree is usually suggestive of a lesion of the temporal or frontal lobe of the brain.

ACALCULIA Difficulty or loss of ability for calculation or mathematical reckoning may occur as part of a motor aphasia or occur independently, it may be due to a lesion of Broca's area or parietal lobe lesion. In amusia, there is a selective loss of ability to produce or understand music or musical sounds. In semantic aphasia, words lose their meaning. In syntactic aphasia, the power of grammatical construction is lost.

GLOBAL APHASIA (Central aphasia) A combination of receptive and expressive aphasia where there is loss of understanding and expression of spoken and written speech. The receptive and productive mechanisms of speech are usually affected in an unequal degree. Spoken speech may be affected more than written speech or vice-versa. In this variety of speech disorder, there is defective formation of inner speech or basic word-schemes. Whilst in pure motor aphasia and nominal aphasia inner speech is formulated and the patient knows what to say but cannot find the right words to say it, in global aphasia he has no conception at all of what to say.

The lesion responsible for central aphasia, although not clearly elucidated, is probably diffuse damage of both temporal (superior temporal convolution) and parietal lobes.

CORTICAL DEAFNESS Deafness due to failure of the central or cortical mechanism of hearing but with its peripheral mechanism (ear, eighth cranial nerve and brain stem centres) intact, as encountered in rare cases of bilateral damage of the temporal lobes, is referred to as cortical deafness. The patient is incapable of hearing any sounds at all.

EXAMINATION FOR APHASIA Before carrying out any special tests for aphasia, it is important to determine (1) the general level of education, literacy and intelligence of the patient, usually on the basis of occupation, attainments and background, (2) whether right-handed or left-handed, (3) whether the hearing and sight of the patient are adequate for testing, (4) whether there is any motor disability of the hand used for writing, and (5) whether the patient is suffering from confusion and if so, in what degree. Confusion results in defective comprehension and formation of verbal symbols, the effect of which is to produce symptoms of dysphasia.

In an investigation of speech disorders, there are four major components of speech (*speech functions*) that must be tested, viz, speaking, listening, reading and writing. Each of these may be impaired through (a) cortical damage, (b) damaged subcortical connections, (c) general impairment of cerebral function, or (d) psychological factors.

The investigation of aphasia should proceed systematically as follows

(1) Speaking (Production or expression of spoken speech)

(a) Spontaneous speech The patient's ability to express himself in speech is observed. Is he unable to speak or is the speech limited to a few words? If able to speak, encouragement to talk may be given in a variety of ways appropriate to the patient ranging from "How are you today?" "Where do you live?" Note incorrect construction, defective grammar, use of wrong words, use of non-existent words Does he speak only under the influence of emotion? Ask him to recite serials such as days of week, months of the year, alphabet, prayer or poem.

(b) Repetition (i) On command letters, syllables, words, short sentences, complex sentences (ii) Compulsive and automatic (echolalia)

(c) Naming objects The patient is asked to name a series of common objects shown to him. Very slight degree of dysphasia may be revealed by this test. The patient may name all objects correctly except one and that a very familiar one such as a pen, although he is able to describe it in terms of its use as "what you write with" The objects shown should be in the following order those easily named being mingled with those more difficult Penny (coin), button, handkerchief, pencil, wrist watch, fountain pen, collar, cigarette case, blotting paper, cuff links, paper, tooth-brush or wastepaper-basket, electric torch, ash tray

(2) Listening (Understanding spoken language) The patient is given a series of verbal commands (i) Put out your tongue (ii) Take hold of my hand. (iii) Touch your left ear (iv) Touch your right ear with your left hand. (v) To explain meaning of simple phrases or sentences.

(3) Reading (a) Reading aloud. The patient is asked to read aloud a paragraph from a book or newspaper A passage should be chosen, the meaning of which the patient would apart from any defect due to dysphasia, is able to understand. A note should be made of any mistakes, including mispronunciation and repetition. With selective impairment he may read letters but not words, or commonly he may read syllables but not letters or words

(b) Written commands A series of written commands should be presented to the patient in order of increasing complexity The command should first be written in legible handwriting. If there is failure, it should be repeated in block capitals The commands are Open your mouth, put out your tongue, put your hand on top of your head, point to your left eye, touch your right ear with your left thumb, when I lift my right hand above my head but not before, pick up a pencil

(4) Writing The patient is given pencil and paper and asked to write his name and address, names of a series of objects shown, his previous occu-

pation, weather, of his own complaints, of recent news in the papers, of the happenings of his daily life. He must be asked to write to dictation or to copy from a book.

5 Associated speech phenomena The ability to understand pantomime and express thoughts in gesture language is tested by asking the patient to reply to questions with a "nod" (for "yes") or a "shake" of the head (for "no") and to signify numbers with fingers. Loss of such ability is referred to as *amimia*, and its derangement as *paramimia*.

## DYSARTHRIA

A disorder of the peripheral mechanism of speech, involving a disturbance of function of the muscles of phonation, articulation or enunciation is referred to as dysarthria. Because of its distinctive aetiology and significance, dysarthria must be clearly distinguished from aphasia. According to site of lesion, dysarthria may be classified aetiologically as follows:

(1) Neuromuscular disorders Facio-scapulo-humeral form of muscular dystrophy or dystrophia myotonia, or myasthenia gravis

(2) Lower motor neurone disease Bell's palsy, acute infective polyneuritis, acute anterior poliomyelitis, motor neurone disease

(3) Upper motor neurone disease Pseudo-bulbar palsy, as in arteriosclerosis (bilateral affection, usually)

(4) Extrapyramidal disease Parkinsonism

(5) Cerebellar disease Multiple sclerosis

(6) Involuntary movements When involving the tongue, face, palate or muscles of respiration, such movements may interfere with articulation as in chorea and athetosis.

Types of dysarthria, Many different varieties of dysarthria have been described, the most important being

(1) SLURRED SPEECH (SLURRING SPEECH) It sounds as if the patient is trying to speak with the mouth full or is intoxicated or inebriated. It is encountered in general paralysis of the insane, pseudo-bulbar palsy and upper brain stem tumours.

(2) STACCATO SPEECH (ATAXIC SPEECH) An intermittent, jerky or explosive form of speech encountered in cerebellar disorders and multiple sclerosis.

(3) SCANNING SPEECH (SYLLABIC SPEECH) A slow and measured variety of speech, with the syllables pronounced clearly and individually, and separated by short pauses. It is typically encountered in disseminated sclerosis and (at times) in lesions of the brain stem or cerebellum.

(4) THICK BULBAR SPEECH Heavy and thick speech, encountered in a case of damage or disease of medullary speech nuclei or cortico-bulbar tracts.

(5) Feeble slurred speech A distant and indistinct form of slurred speech, encountered in cases of myopathy and myasthenia gravis.

(6) STAMMERING SPEECH A common variety of speech defect of uncertain causations and at times, remediable.



(7) ANARTHRIA. A severe form of dysarthria, with no sounds emitted from the mouth, as in rare cases of bulbar or pseudo bulbar paralysis

(8) RIGID DYSARTHRIA. A monotonous speech with sentences starting and stopping abruptly, commonly seen in Parkinsonism

(9) LOWER MOTOR NEURONE DYSARTHRIA, e.g. facial, tongue, or palatal paralysis (nasal dysarthria) Speech is preserved but individual words and sounds cause difficulty

(10) DYSARTHRIA ASSOCIATED WITH DYSPHASIC STATES Some degree of dysarthria may be observed in presence of expressive dysphasia, and may rarely accompany lesions producing severe apraxia of muscles of articulation Association of dysphasia and dysarthria may also occur with thrombosis of internal carotid artery It is important to bear in mind that in presence of mixed defects of speech, dysphasia is the most important localising factor

Miscellaneous forms of speech disorders The best known are the following

*Echolalia* Involuntary repetition of words and phrase spoken by someone else, unintentionally and without understanding the meaning It may be seen in lesions of the temporo-parietal region

*Mirror speech* A peculiar defect of speech, where syllables or words are pronounced backwards

*Oesophageal and laryngeal speech* These are two varieties of post-laryngectomy speech disorders the former being associated with air-swallowing and eructational sounds, whilst the latter is caused by the artificial larynx

*Lalling speech* (Baby speech) When a grown up patient speaks "baby-fashion" with a dropping out of all difficult consonants, the condition is suggestive of congenital auditory imperception or inborn inability to understand the meanings of sounds When associated with words of the patient's own fabrication, the condition is referred to as *idioglossia*

## APRAXIA

Apraxia is defined as the inability to perform well-organized voluntary movement correctly in absence of significant impairment of motor, sensory and co-ordinative functions The performance of complex, purposive, skilled acts requires three steps (i) Development of the idea of what is required and retaining this concept till the act is completed (ii) Formulation of a plan to accomplish the act This requires a knowledge of location of one's body and body parts and the relationship of the surroundings (iii) Putting into effect the details of the plan with the help of skilled movements Apraxia may result from failure of any one of these steps The types of apraxia are

*Ideational apraxia* It is due to failure of step (i) and results in a loss to understand the idea, or there is defective formulation of the method of carrying out the complex act

*Ideomotor (ideokinetic) apraxia* It results from failure to execute the idea (step ii) This is common in hemiplegic patients but the disability affects the normal extremities

*Kinetic apraxia* It is due to failure of the third step There is inability to make fine skilled movements such as writing or piano-playing This phenomena may result from a lesion of the prefrontal motor cortex

Apraxia is produced by lesions which interrupt the connections between the parietal cortex of the dominant hemisphere to the motor cortex of both hemispheres Thus (a) a lesion involving the supramarginal gyrus of the dominant parietal lobe can produce bilateral apraxia, (b) a lesion in the dominant hemisphere disconnecting the parietal cortex from the motor area can produce right sided apraxia, while (c) a lesion of the corpus callosum, which interrupts the fibres connecting the parietal cortex to the opposite side, can produce left sided apraxia

EXAMINATION FOR APRAXIA It is important as in examination for dysphasia to ascertain the presence of confusion and assess the degree if present

History Does the patient complain of incapacity to carry out habitual or skilled movements? Does he find difficulty in shaving, dressing? Has he been observed to have such incapacities? If so, is he aware of them? Does the disorder relate to simple habitual movements, or to some highly skilled or complex acts? Does it affect one limb only or all?

Commands which the patient is asked to carry out are

(i) Ask him to show how to use a common object—comb, toothbrush, pair of scissors

(ii) Patient is given a packet of cigarettes and a box of matches and asked to light a cigarette, or he is given a sheet of paper and envelope and asked to fold the paper and place it in and close the envelope

(iii) Make a fist, clasp your hands, put out your tongue, close your eyes

(iv) Wink, salute (or namaste)

(v) Pretend to knock at a door, play a piano or drive a car

(vi) Construction Drawing simple geometrical figures such as a triangle or square on paper Arranging matchsticks to construct figures in imitation of those made by the examiner

## AGNOSIA

Agnosia is failure to recognize familiar objects or sounds when the sense by which it is normally recognized remains intact

### EXAMINATION FOR AGNOSIA

1 Tactile recognition of common objects (Stereognosis) (a) Amorphognosis Inability to distinguish size and shape (b) Anhylognosia Fail-

ure to recognize differences of density, weight and roughness (c) Tactile asymbolia Failure to recognize objects by touch although (a) and (b) are not responsible

Tactile agnosia results from lesions in the contralateral supramarginal gyrus, (parietal cortex)

2 *Auditory recognition* First determine if hearing in both ears is normal Ask patient to close his eyes and to identify sounds made by crumpling of paper or jingling of keys or coins

Auditory agnosia results from lesion in the posterior part of the temporal lobe of the dominant hemisphere

3 *Visual recognition* (a) Of objects (b) Of colour (c) Of faces (d) Appreciating the meaning of a picture as a whole (for simultagnosia) (e) Visual spatial relationships Ask patient to look at an object and then with eyes closed to point in that direction (f) To divide a straight line in the middle (g) To draw a clock-face (h) To draw common objects from memory (i) To describe and copy objects in pictures (j) Topographical memory to describe a familiar route from memory

In case of visual agnosia for objects and colours the lesion is in the second and third occipital gyri of the dominant hemisphere

4 *Recognition of body schema* (for somatopagnosia) To find out if there is disturbance in recognition of patient's own body parts, ask patient to (a) Identify parts of his body (b) Identify parts of the observer's body (c) Recognition of sides (d) To identify digits of his own hand and of the observer's hand (e) Unawareness of the existence of any part of his own body (f) Denial of illness (for anosognosia), e.g. hemiplegia, deafness, blindness

## THE CRANIAL NERVES

### THE OLFACTORY NERVE

*Anatomy* The filaments of the first cranial nerve arise within the olfactory mucous membrane of the upper part of the nasal fossa, and end in the olfactory bulb, underneath the frontal lobe. A fresh relay of fibres, forming the olfactory tract, leads to the cortical centre for smell in the piriform lobe in the anterior end of the uncus and the hippocampal gyrus. All these structures are associated with the sense of smell.

Since disease of the mucous membrane of the nose can interfere with olfactory function, examination of the first cranial nerve is of limited value.

*Test of function* The *sense of smell* is tested in each nostril separately, one being closed with digital pressure while testing. Small bottles containing smelling substances, such as oil of peppermint, oil of cloves, coffee and tincture of asafoetida, are in turn held close to the nostril, the patient being asked

to inhale with the mouth closed and try to recognize the odoriferous substance. Pungent substances, such as ammonia and acetic acid, should not be used because they are likely to stimulate trigeminal nerve endings and irritate the nose even when the sense of smell is lost. Local obstruction or disease of the nose may interfere with the test.

### Abnormalities of smell

**ANOSMIA** Loss of olfactory sense (*anosmia*) or diminished sense of smell (*hyposmia*), barring local nasal conditions, like atrophic rhinitis, may be due to (1) Fractured floor of the anterior cranial fossa, with damage to the olfactory fibres (2) Compression of olfactory tract by tumour, such as an olfactory groove meningioma, or by a distended cerebral hemisphere, as in obstructive hydrocephalus (3) Meningitis purulent, syphilitic or tuberculous (4) Atrophy or degeneration of olfactory nerves, as in tabes dorsalis (5) Hysterical. In organic anosmia, ammonia can be identified but not coffee.

**PAROSMIA** A perverted sense of smell may be noted after a head injury, the patient being unable to interpret the right nature of an odour. For instance, oil of cloves may be mistaken by the patient for asafoetida.

**HYPEROSMIA** Lowered threshold for odours may be encountered in Addison's disease and mucoviscidosis.

**HALLUCINATION OF SMELL** An irritative lesion of the olfactory apparatus, particularly when involving the temporal lobe, may produce *uncinate fits* or attacks, characterized by hallucinations of smell or taste and associated with a "dream-like state". Such attacks are indicative of a focal lesion in the hippocampal gyrus, or (rarely) the "olfactory aura" of epileptic states.

### THE OPTIC NERVE

**Anatomy** The optic or second cranial nerve originates within the cellular layer of the retina and enters the cranial cavity through the optic foramen in the sphenoid bone. The two optic nerves emerge to form a semi-decussation in the chiasma. Whilst the fibres from the inner halves of the two retinae decussate, those from the outer halves continue straight without crossing. From the chiasma, the optic tracts continue backwards to end in the primary optic centres or cerebral ganglia (external geniculate body, pulvinar of the optic thalamus and anterior corpus quadrigeminum) on either side. From the external geniculate body arises a second set of nerve fibres, the optic radiation, which, after traversing the posterior limb of the internal capsule, spreads backwards in a fanwise manner to terminate within the visual cortex or chief visual centre, around the calcarine fissure of the occipital lobe. The right half of the field of vision is represented in the right cerebral cortex, and the left half in the left cortex.

**Tests of function.** The following four features must be investigated routinely in the examination of the optic nerve (1) acuity of vision, (2) field of vision

(3) colour sense, and (4) examination of fundus Each eye must be investigated individually, after ruling out errors of refraction and opacities of the media.

(1) Acuity of vision The visual acuity of each eye is tested separately for distant and near vision

(a) PERCEPTION OF LIGHT When visual acuity is markedly affected, the patient may be totally blind or unable to distinguish light from darkness Either the light from a torch is intermittently turned on and off, or the patient's eye alternately covered and uncovered, whilst facing a lamp, for this purpose

(b) HAND MOVEMENTS AND FINGER COUNTING TESTS The patient may be able to perceive hand movements in front of the eyes, even when unable to count fingers Different numbers of fingers of the examiner's right hand are held before the patient's eye, at varying distances, and the patient asked to count them

(c) SNELLEN'S TEST TYPES *Distant vision* can be tested in patients with minor derangements of visual acuity, with the aid of a Snellen's chart, placed 6 metres (or 20 feet) away, on level with the eyes of the patient The number beside each line indicates the number of feet or metres at which the letters can be read by one with normal vision. The *acuity of vision* is expressed as a fraction, the numerator corresponding to the distance from the patient to the chart (6 metres) and the denominator indicating the distance at which the smallest row of letters read by the patient should be read by the normal eye If the patient's eyesight is normal, he will read the smallest type at 6 metres and the acuity of vision will equal 6/6 If at 6 metres the patient can only read the largest type, type which he should be able to read at 60 metres, the visual acuity will be 6/60

(d) JAEGER'S TEST TYPES *Near vision* can be tested with the aid of the Jaeger test chart or card, held 14 inches from the patient's eyes

The number under the test chart indicates the distance in inches at which the letters can be read with normal vision The line of smallest letters that the patient can read is determined When visual acuity and accommodation are normal, the patient is able to read the smallest type with each eye separately, at a distance of about 30 cm

DISTURBANCES OF VISUAL ACUITY *Amblyopia* Defective acuity of vision, not amounting to total blindness (*amaurosis*), is referred to as amblyopia As a rule, amblyopia is bilateral and transient and due to drugs, or toxins (e.g. tobacco, ethyl or methyl alcohol, ergot, quinine, salicylate, lead, arsenic and digitalis), diabetes, uraemia, migraine, haemorrhage, or lesion of the visual cortex

Amaurosis (Complete blindness) This may be gradual or sudden in onset, unilateral or bilateral, transient or persistent. Gradual blindness may be due to inflammatory conditions of the eye, optic atrophy or glaucoma and is usually unilateral to begin with. Sudden blindness may be unilateral or bilateral, transient or persistent, and due to a variety of causes, such as detachment of retina, acute glaucoma, spasm or embolism of central retinal artery, thrombosis of central retinal vein, vitreous haemorrhage, retrobulbar neuritis, migraine, tobacco or alcohol poisoning, uraemia, hypoglycaemia, injury to optic nerve or hysteria.

Hemeralopia (Day blindness) A common symptom of albinism or tobacco amblyopia, hemeralopia or defective vision during daytime or in bright light may be due to fatigue of the retina and pupillary contraction to light resulting in a cutting out of the peripheral zones of the retina.

Nyctalopia (Night blindness) Loss of vision at night or in darkness may occur in Vitamin A deficiency, retinitis pigmentosa or quinine poisoning.

Teichopsias (Fortification figures) Coloured, zigzag, glittering figures or scotomas, referred to as fortification spectra may occur transitorily during attacks of ophthalmic migraine and in case of occipital tumour.

(2) Field of vision The field of vision represents that portion of space, in which other objects are visible when fixing the gaze on a particular object in any direction. For instance, when looking at a particular tree on the right, the various other items in the vicinity of the tree that the patient observe automatically and at the same time, collectively, represent his field of visions. For each gaze or act of visual fixation, there is a visual field. The peripheral limits or margins of the visual field, for a given patient, are usually constant, and depend partly on the state of his eyes and partly on the delimitation of the visual field by the nose, cheek and orbit of the patient.

Since lesions affecting the optic pathway can cause specific types of visual field defects, a routine mapping out of the visual field (perimetry) is an essential and important part of any neurological examination. It is frequently possible to determine the site or nature of a neurological lesion by recourse to perimetry. Several methods of investigation are available for this purpose.

(a) CONFRONTATION METHODS There are two methods (both only roughly accurate) available for this purpose.

(1) The examiner stands or sits opposite the patient at a distance of 3 feet. Each eye is tested separately (Fig 13 2A). To test the patient's right eye,

he is asked to cover his left eye with a piece of paper held in his left hand and keep gazing at the examiner's left eye. The examiner closes his right eye in similar manner and looks with his left eye steadily at the patient's right eye. The four *quadrants* of vision, namely the upper and lower, nasal and temporal, are tested separately, by moving a finger (or two) of one of the hands, along a plane midway between the examiner and the patient, inwards, from various points at the periphery towards the centre. The patient is asked to say "yes" when he begins to see the examiner's mobile finger. This normally coincides with the point at which the examiner himself catches sight of the moving finger, presuming, of course, that the examiner's own field of vision is normal.

(ii) In the other method, the examiner stands or sits directly in front of the patient, with both eyes open and moves or wiggles the fingers of both hands simultaneously (Fig 13 2B), from the outer limits of the visual field inwards. This will detect the phenomenon of visual inattention common in parietal disorders when though the fields are normal, only one object is appreciated when both are moved simultaneously.

In unco-operative or mentally deranged patients, an observation of optically induced eye movements may prove of value. Some shiny or attractive object is brought within the periphery of the visual field from different directions. If the patient turns his eyes in the direction of the object, the visual field in that particular sector is likely to be intact or unaffected.

The patient's field of vision for a stationary object (such as an immobile pencil or finger of the examiner's hand) is frequently different from and more limited than for a mobile object (as described above).

In the case of children or difficult patients, resort may be had to the defensive blinking reaction (menace reflex) for mapping out visual fields. The hand or finger of the examiner's hand is moved suddenly and in a threatening manner, towards the patient's eye, from different directions, and the eye watched closely for a blinking reaction.

(b) INSTRUMENTAL METHODS. This may be accomplished either with the aid of a mechanical instrument called the perimeter, many different models of which are available, or with a Bjerrum's screen, the latter being more useful for investigating central portions of the visual field. For accurate exploration of the peripheral portions of the visual field, however, the perimeter remains invaluable.

Interpretation (Fig 13 3). Optic fibres from the inner half of each retina cross in the chiasma, whilst fibres from the outer half remain uncrossed. Lesions affecting the optic nerve, anterior to the chiasma, therefore, produce

unilateral impairments of vision, whilst lesions of the chiasma produce bilateral visual field defects. The various fields of vision are named (*nasal* or *temporal*) according to their proximity to the nose or temple

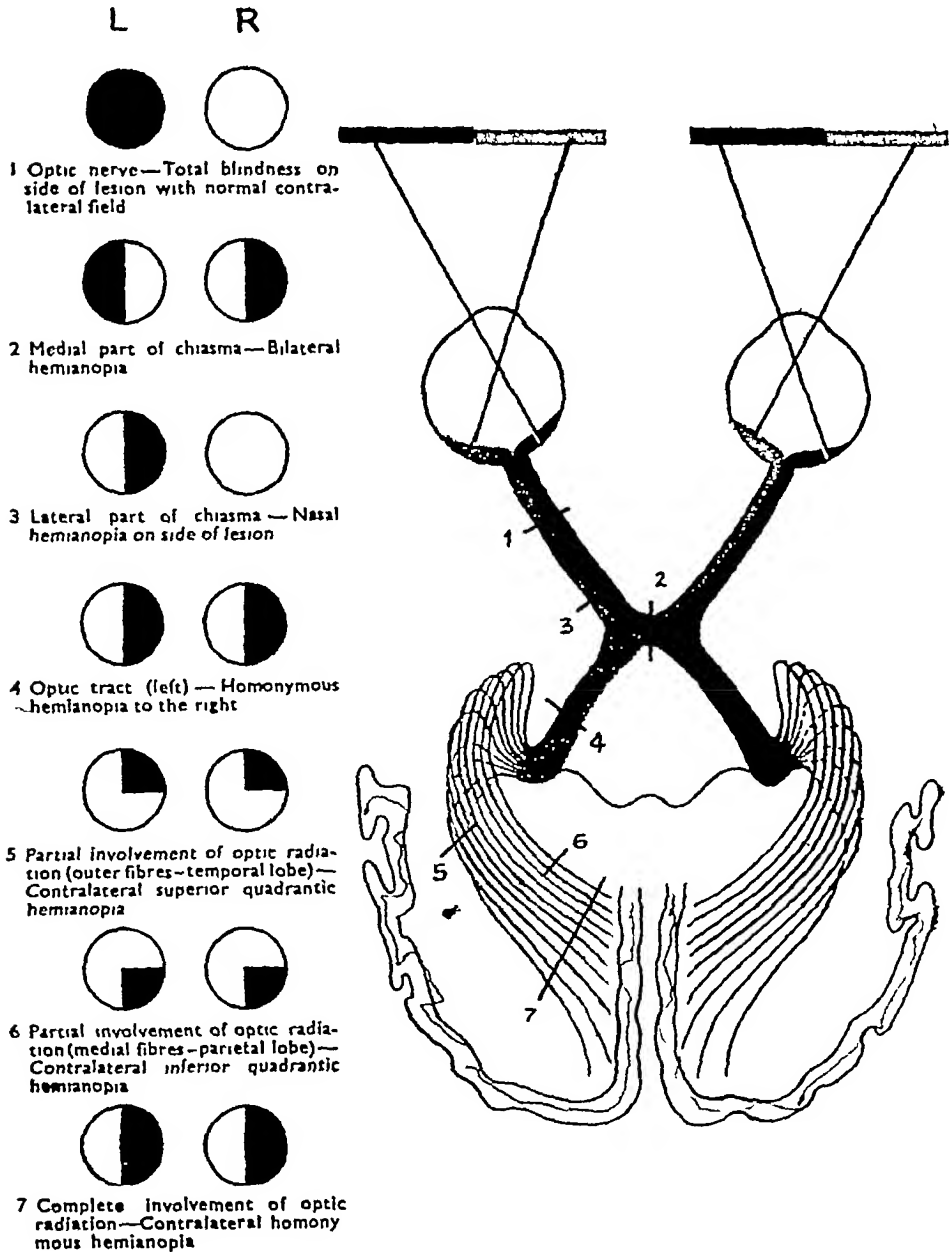


Fig 13.3 The abnormal visual field. Schematic representation of the visual pathways showing sites of interruption of nerve fibres and various abnormal field defects thus produced



VISUAL FIELD DEFECTS These may be of many types. A correct interpretation of the type of defect may facilitate clinical diagnosis.

(a) Peripheral constriction A concentric diminution of the visual field, all round the periphery, may be due to hysteria, optic atrophy, bilateral involvement of cortical centres, or retinal disease.

(b) Scotoma A defect within the visual field is termed a scotoma. The latter may be central, paracentral or peripheral, unilateral or bilateral, positive or negative. The "blind spot" may be referred to as a physiological scotoma. The term positive scotoma is used, when the patient sees a dark spot in the visual field, which does not correspond to any real object outside the eye. A negative scotoma (scotoma proper) refers to an abnormal blind area or defect in the visual field, an area in which the patient is unable to observe any external object. A negative scotoma is called absolute, when perception of light is entirely lost in the affected area, and relative when partially lost or diminished. A scotoma in the peripheral portion of the field does not, as a rule, affect vision appreciably. A central scotoma, on the other hand, can prove a great hindrance when unilateral, central scotoma is usually due to local eye disease, optic or retrobulbar neuritis, or multiple sclerosis. Bilateral central scotoma may be toxic in origin (tobacco or alcohol poisoning) or secondary to a lesion of the visual centre in the cortex.

(c) Hemianopsia (Hemianopia) Blindness or loss of vision in one-half of the visual field, from non-retinal causes, is called hemianopsia. It may be unilateral or bilateral. When the upper (superior) or lower half (inferior) of the visual field is affected, it is described as altitudinal hemianopsia. When the lateral or medial half of the visual field is affected, as is usually the case, the condition is called lateral hemianopsia. Depending on whether similar halves (right halves or left halves, i.e. temporal half of one side and nasal half of the other) or dissimilar halves (outer halves or inner halves) of the two fields of vision are affected, the condition is described as homonymous or heteronymous hemianopsia. Blindness may affect the nasal or inner halves of the visual fields (binasal hemianopsia), as in rare instances of bilateral disease involving the optic chiasma. Unilateral nasal hemianopsia occurs in case of compression of the lateral part of the chiasma by an aneurysm of the internal carotid artery or an atherosclerotic internal carotid artery. Loss of the temporal or outer halves of vision (bitemporal hemianopsia) occurs in lesions of central part of the chiasma containing the decussating fibres. The commonest cause of such a lesion is a pituitary tumour. When pressure is exerted on the chiasma from below, the lowermost fibres of the chiasma (which arise from the lower part of the nasal halves of the retinae) are compressed and the first evidence may be a superior bitemporal quadrantanopia. This may gradually extend to involve the entire temporal half of the field first on one side and then the other. The initial defect may be only for colours.

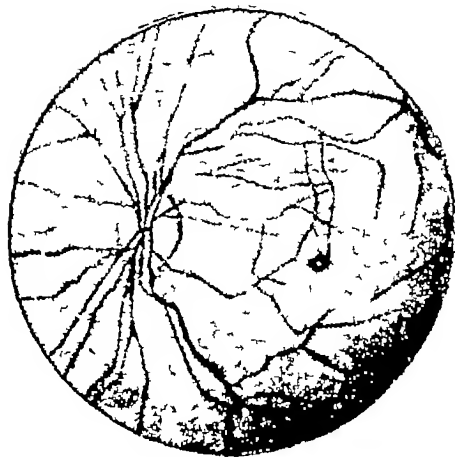


Fig 134 Normal fundus

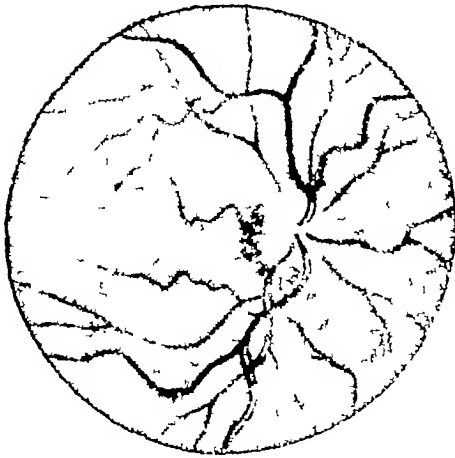


Fig 135 Papilloedema

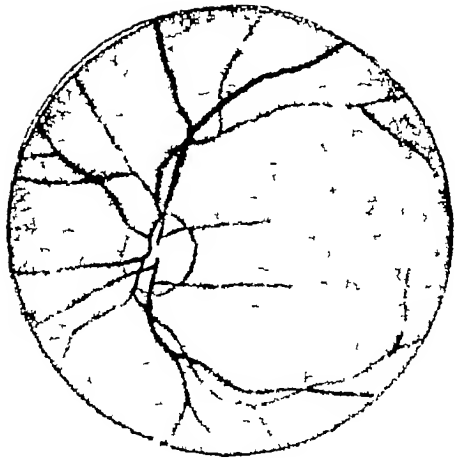


Fig 136 Optic atrophy



Complete homonymous hemianopia occurs in lesions of optic tract or optic radiation. Certain points help to differentiate between the two. The fibres of the radiation spread out like a fan while fibres of the optic tract are in a compact bundle. Lesions of the radiation therefore more often produce quadrantic or incomplete defects. Lesions of the optic tract also involve the fibres which are involved in the pupillary reflex. Thus, if light is shone in the hemianopic field of vision, the pupillary reflex is absent if the lesion involves optic tract, the pupillary reflex is spared if the lesion is in the optic radiation. Macular sparing occurs more often in lesions of optic radiation.

(3) Colour vision Colour blindness in a patient can be usually discovered with the aid of specially coloured cards or skeins of wool (Holmgren's wools). Three main varieties of colour blindness are recognizable red-green blindness, yellow-blue blindness and total colour blindness, in order of importance.

The extent of the visual field is not the same for different colours, it is maximal for white and minimal for green. Colour vision may be affected in lesions of the cortex as well as in hysteria.

(4) Ophthalmoscopic examination (Fundoscopy, retinoscopy) Examination of the fundus is an absolute essential in every neurological examination. The examiner stands or sits on one side facing the patient. The ophthalmoscope is brought directly in front of the patient's eye, as close to it as possible. The patient is asked to look at the opposite wall or directly over the shoulder of the examiner. When the patient and examiner are both emmetropic, a clear view of the fundus can be obtained by looking through the sight hole without the aid of any lens. The examiner holds the ophthalmoscope in his right hand, and uses the right eye for examining the patient's right eye. The technique is then reversed for the left eye.

The normal fundus (Fig 13 4) presents an orange-red surface, over which can be distinguished the optic disc, retinal blood vessels and macula. The optic disc, which represents the entrance of the optic nerve into the retina, is usually circular, light pink in colour (the lateral half being paler than the medial half), and has well-defined margins. In the centre of the disc is a funnel-shaped depression, whiter than the rest of the disc, known as the physiological cup.

Retinal blood vessels Arteries of the retina can be readily distinguished from veins by their smaller calibre, bright red colour and straight course. They display bright reflections along their centres. Arteries and veins follow approximately the same course.

The retina itself is transparent, the colour of the fundus being derived from the choroidal vessels. The macula, which is physiologically the most important part of the fundus, is situated on the temporal or outer side of the optic disc and is devoid of visible vessels and is somewhat darker in colour than the rest of the fundus.

**ABNORMAL TYPES OF FUNDI** Three types of abnormal fundi, namely papilloedema (or choled disc), optic atrophy and optic neuritis, are of particular significance in neurology

Papilloedema (Fig. 13.5) Oedema of the optic nerve-head, when of non-inflammatory origin, is usually referred to as papilloedema. It is most commonly observed in association with increased intracranial tension, secondary to intracranial tumour or meningitis, or with systemic hypertension. The diagnostic features of such a fundus are swelling and redness of the disc, blurring of disc margins, poorly defined physiological cup, engorgement and tortuosity of veins, narrowing of arteries, and relatively insignificant impairment of visual acuity.

Optic atrophy (Fig. 13.6), which is due to degeneration of optic nerve fibres, may be either primary (or simple), as observed characteristically in tuberculosis, or secondary, usually arising as the result of long-standing papillitis or papilloedema. The characteristic ophthalmoscopic finding is a pale grey or white disc, somewhat smaller in size than normal, and with well-defined, sharp margins. The lamina cribrosa is clearly seen, and the retinal vessels appear normal.

Foster-Kennedy syndrome A tumour in the posterior inferior frontal region can cause optic atrophy on one side by compressing the optic nerve, and papilloedema on the other by obstructing venous return or causing raised intracranial pressure.

Optic neuritis This is a term used to describe involvement of the optic nerve as the result of inflammation or demyelination. The fundus shows hyperaemia of the disc with swelling, blurred disc margins, and a partially or totally obscured physiological cup. Exudates and haemorrhages may be observed close to the disc. The arteries are usually thin and the veins dilated and tortuous. Loss of visual acuity is usually out of proportion to the disc changes, and a scotoma may be present.

## OCCULOMOTOR, TROCHLEAR AND ABDUCENT NERVES

It is customary to examine these three cranial nerves, which innervate muscles concerned with ocular movements, together

**Anatomy** The oculomotor nerve arises from a nucleus in the floor of the aqueduct of Sirius, extending from the caudal end of the third to the ventral end of the fourth ventricle.

The nucleus is made up of—(1) a large paired lateral nuclear mass from which arise the fibres supplying various ocular muscles. (2) A median unpaired nucleus (nucleus of Parinaud) which is the centre for convergence and accommodation. (3) Edinger Westphal nucleus, which is paired, and constitutes the parasympathetic nucleus supplying fibres to the ciliary muscle of the pupil.

nerve radicles pass through the substance of the red nucleus and substantia nigra the interpeduncular fossa. At the base of the brain, the nerve, first situated in the cranial fossa, extends forwards to pierce the dura in the middle cranial fossa, passes the cavernous sinus, and enters the orbit through the superior orbital fissure, to the muscles of the eyeball, except the superior oblique and lateral rectus muscles.

The trochlear nerve has its nucleus, just below that of the oculomotor nerve, within the grey matter of the floor of the aqueduct of Sylvius. After entering the anterior medullary velum, the nerve fibres decussate and emerge on the dorsal aspect of the midbrain, just below the inferior colliculus. At the base of the brain, the nerve emerges on one or other side, winds round the cerebral peduncle above the pons and pierces the dura to enter the cavernous sinus, lying below the third cranial nerve. The nerve then enters the orbit, through the superior orbital fissure, to supply the superior oblique muscle.

The abducent nerve has its nucleus in the floor of the fourth ventricle, within the loop formed by the facial nerve near its origin. The fibres from the nucleus pass forwards and downwards to emerge in a groove between the pons and the medulla. The nerve pierces the dura opposite the dorsum sella, runs under the petrosphenoidal ligament, and enters the cavernous sinus. After traversing the sinus, the nerve enters the orbit, through the superior orbital fissure, and innervates the external rectus muscle. Its long course makes it particularly vulnerable to the effects of raised intracranial tension.

Whilst the fourth cranial nerve supplies the superior oblique muscle and the sixth the external rectus, the third nerve innervates the remaining ocular muscles, as well as the ciliary muscle and constrictor muscle of the iris.

### Tests of function

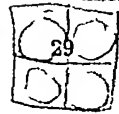
(1) The eyelids Look for drooping of the upper eyelid (ptosis), by observing how much of the white of the eye is exposed. In case of doubt, the patient is asked to look up. In true ptosis, the patient cannot raise the eyelid to its full extent, except by contraction of the frontalis muscle, which compensates for the ptosis. If the frontalis is fixed by the examiner's finger, so as to prevent this compensatory action from occurring, the patient is unable to raise his eyelid. In severe ptosis of long standing, owing to the continuous contraction of the frontalis muscle (which enables the patient to raise his eyes from the ground), the face tends to assume a peculiar expression, the head being, at times, held in a retroflexed position, as in case of ophthalmoplegia or tabes dorsalis.

Ptosis may be due to paralysis of the third nerve, paralysis of cervical sympathetic, myasthenia gravis, ocular myopathy, inflammatory lesions of the conjunctiva or trachoma. Congenital ptosis is usually bilateral. The difference in the palpebral fissure in Horner's syndrome disappears upon upward gaze, while that in oculomotor palsy is accentuated.

Ptosis may be due to paralysis of the levator palpebrae superioris as a result of third nerve lesion, or to weakness of the tarsal muscles due to a sympathetic lesion. In the latter case the lid can be raised voluntarily but in the former, the frontal muscles contract to overcome the drooping and there may be permanent wrinkling of the forehead.

(2) Position and appearance of the eyeballs at rest. (a) Involuntary deviation There may be a tendency to look continuously to one or other side, upwards, or downwards (conjugate deviation). In cerebellar or quadrigeminal lesions the ipsilateral eye looks downwards and inwards, whilst the other eye is directed upwards and outwards (skew deviation).

(b) Exophthalmos A unilateral protrusion of the eyeball may be due to unilateral myopia (of high degree), primary tumours of the optic nerve or its



sheath, diseases of the paranasal air sinuses, toxic goitre, inflammatory lesion such as retrobulbar abscess or cellulitis, thrombophlebitis of orbital veins, thrombosis of cavernous sinus, periostitis, orbital exostosis, fractured orbit, laceration or rupture of tissues within the orbit, pulsating exophthalmos, carotid-cavernous aneurysm, retrobulbar haemorrhage (as in whooping cough), or haemopoietic disease (like Hodgkin's disease, lymphosarcoma, chloroma or scurvy) *Bilateral* exophthalmos commonly occurs in *thyrotoxicosis*

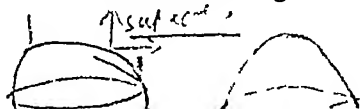
(c) *Enophthalmos* A *unilateral* retraction of the eyeball or enophthalmos may be due to trauma, paralysis of cervical sympathetic (*Horner's syndrome*, Fig 13 7), diminution of orbital contents through operation, spontaneous subsidence of a pulsating exophthalmos, facial hemiatrophy, or *congenital* anomaly. The eyeballs appear "sunken" in case of *dehydration* ✓

(3) *Ocular movements* The ocular movements consist of (a) *lateral deviation*, i.e. adduction and abduction, (b) vertical deviation i.e. elevation and depression, (c) diagonal movements at intermediate angles and (d) rotation around an imaginary horizontal anteroposterior axis passing through the centre of the pupil. Normally the two eyes move *harmoniously keeping their axes parallel* to each other. These are called *conjugate movements*. Conjugate movements consist of lateral deviation to right or left, elevation, depression and deviation at intermediate angles. Convergence is a special type of conjugate movement consisting of conjugate adduction ✓

**OCULAR MUSCLES** The extrinsic ocular muscles consist of four recti, superior, inferior, medial and lateral, and two obliques, superior and inferior, in addition to the levator palpebrae superioris. The eyeball is a spherical structure which can rotate around three different axes passing through its centre. The lateral and medial recti act in only one plane and produce a pure movement of abduction or adduction. All other muscles act in more than one plane. Thus superior rectus, in addition to being elevator, is also adductor and internal rotator of the eyeball. The inferior rectus is depressor adductor and external rotator. The superior oblique is a depressor, abductor and internal rotator, while inferior oblique is elevator, external rotator and abductor.

When the eye is abducted by 23 degrees, the superior and inferior recti become pure elevators and depressors. When the eye is turned inwards (adducted) the obliques tend to become pure elevators or depressors.

While elevating the eye, superior rectus is helped by inferior oblique, their horizontal and rotating components cancelling each other. Similarly while depressing the eye, the inferior rectus is helped by superior oblique, their horizontal and rotating components cancelling each other.



## EXAMINATION OF OCULAR MOVEMENTS

(a) Movements in response to command Keeping the patient's head steady, he is asked to look in various directions and the conjugate and individual ocular movements noticed

(b) "Follow" movements Keeping the patient's head steady, he or she is asked to follow, with his eyes, the tip of the examiner's finger. Movements in various directions are noted. It is to be noted that "follow" movement differs from "command" movement in that it involves fixation of eyes on an object. It is a reflex movement, the pathway of which includes occipital cortex. In certain lesions of central nervous system, voluntary (command) movement may be lost and yet "follow" movement may be preserved (vide infra)

(c) Reflex movements should be tested since their assessment can provide useful information about brain-stem nuclear function. Reflex ocular movements can be elicited by (i) passive movements of head in an unconscious patient i.e. Doll's eye manoeuvre, (ii) stimulation of the labyrinth, as in the caloric test

Doll's eye movements If the patient is too drowsy to carry out voluntary or following eye movements, the doll's head phenomenon can be employed. This depends on the normal vestibular reflex mechanism which enables ocular fixation to be maintained despite head movement and is based on the fact that with head turning there is a delayed proprioceptive stimulus to move the eye in the direction of movement. In the absence of damage to the brain stem, if the head is passively rotated in a drowsy subject, the eyes remain fixed for a while and then there is conjugate deviation of the eyes to the opposite side. Neck flexion produces upgaze, and extension downgaze.

Caloric stimulation In deep coma of whatever cause, doll's head manoeuvre can be difficult to elicit. In such a case, the effect of caloric stimulation can be used. The external auditory canal is irrigated with 20-40 ml ice-cold water drawn into a syringe. After a brief delay, forced conjugate gaze occurs towards the irrigated ear. Brain stem dysfunction may be revealed by loss of gaze to one side (pontine lesion), or by failure of movement in the adducting eye (lesion in medial longitudinal fasciculus).

The neuromuscular set up which governs and carries out the ocular movements consists of (a) Supranuclear centres and pathways (b) Nuclei of the third, fourth and sixth nerve in the brain stem (c) Internuclear connecting pathways (d) Peripheral nerves i.e. 3rd, 4th and 6th cranial nerves, and (e) ocular muscles.

The anatomy of the nerve nuclei and the course of the nerves has already been described.

Supranuclear centres and pathways The centre for conjugate lateral deviation is situated in the posterior part of the middle frontal gyrus. The fibres arising from this area pass through the corona radiata, genu of the internal capsule and the cerebral peduncle. They



decussate in the midbrain and pass downwards to supply the brainstem nerve nuclei of both sides. Stimulation of the frontal centre or its pathway produces conjugate deviation of eyes to the opposite side. The centre for conjugate vertical movements is not clearly localised. It probably lies in the middle frontal gyrus. The fibres have a course similar to that of fibres for lateral deviation. Fibres concerned with conjugate elevation decussate at a higher level in the midbrain than those concerned with conjugate depression. The pathway for convergence is not completely known. As it involves fixation of vision on an object, the pathway is believed to involve the occipital cortex and the middle frontal gyrus.

*Internuclear connections* As the conjugate movements involve both the eyes, the nuclei of the 3rd, 4th and 6th nerves on both sides are connected to each other. These connecting fibres form a bundle called medial longitudinal fasciculus. It also contains fibres connecting the vestibular nuclei to the ocular nerve nuclei.

**ABNORMAL OCULAR MOVEMENTS** Loss or abnormality of ocular movements (ophthalmoplegia) can occur if a lesion of the central nervous system involves any of the structures described above. Ophthalmoplegias can be accordingly classified as (1) Supranuclear palsies, which produce weakness or loss of conjugate movements. (2) Internuclear ophthalmoplegia, of which the predominant features are nystagmus and dissociation of conjugate movements (vide infra). (3) Nuclear ophthalmoplegia. (4) Individual nerve palsies. (5) Ocular myopathies.

In case of the last three groups, the abnormality depends upon the site of lesion and the structures involved, the common features being (a) defective ocular movement, (b) squint and (c) diplopia.

**ABNORMALITIES OF CONJUGATE DEVIATION** An irritative lesion of the frontal cortex causes conjugate deviation of the eyes to the opposite side. A paralytic lesion of the frontal centre or its pathway causes paralysis of conjugate gaze to the opposite side. The unopposed action of the normal side then produces conjugate deviation to the side of the lesion. In case of brainstem lesions, an irritative lesion produces conjugate deviation to the same side. A paralytic lesion of the brainstem produces weakness of lateral gaze towards the site of lesion and consequent spasmodic deviation of eyes to the opposite side.

Lesions in the upper brainstem produce weakness of upward gaze. Paralysis of convergence is rare but known to occur in Parkinsonism due to encephalitis lethargica.

*Internuclear ophthalmoplegia* The lesions of medial longitudinal fasciculus produce dissociation of conjugate movements. In the anterior internuclear ophthalmoplegia, when lateral gaze is attempted, there is weakness of the adducting eye and nystagmus of the abducting eye. However the adducting eye moves normally on convergence, which differentiates it from isolated medial rectus paralysis. In posteroinferior internuclear ophthalmoplegia, ipsilateral abduction is weak, however the eye abducts normally on caloric

stimulation, which differentiates it from isolated 6th nerve palsy. Internuclear ophthalmoplegias occur in multiple sclerosis, vascular lesions, encephalitis and tumours.

**Clinical features of individual nerve palsies** As stated earlier, an injury or lesion affecting the third, fourth or sixth cranial nerve or its nucleus may produce squint, diplopia, defective eyeball movements, pupillary abnormalities, protrusion of eyeball or ptosis. Diagnosis of the lesion can be facilitated by keeping in mind the characteristic features of each type of nerve paralysis.

**THIRD NERVE PARALYSIS** (Fig 13.5) (a) There is ptosis, with loss of ability to open the eye, the ptosis being usually much more pronounced than in case of cervical sympathetic paralysis. In partial palsy, ptosis may be the sole indicator of a third nerve affection, the other muscles being (rarely) spared. (b) The eyeball is deviated outward and slightly downward (divergent strabismus). (c) The pupil is dilated and does not react to light, the power of accommodation is lost. Thus, ptosis with a larger pupil, which reacts sluggishly to light, suggests an oculomotor lesion, while if the pupil is small, a lesion of the sympathetic should be suspected.

**FOURTH NERVE PARALYSIS** (a) Impaired ability to turn the eye downward and outward (due to paralysis of the superior oblique muscle). On attempting to look downward, the eyeball turns inward (due to inferior rectus action). (b) A squint is rare. (c) Diplopia occurs on looking downward, the images being uncrossed. It is usually prevented by tilting the head forwards and towards the normal eye.

**SIXTH NERVE PARALYSIS** (a) A squint is often present, with the affected eyeball deviated inwards (convergent strabismus). (b) Diplopia is present in practically all directions of movement of the eyes (except on gazing away from the side of the lesion). (c) Impaired ability to turn the affected eye outward (Fig 13.9).

**SQUINT (Strabismus)** By squint is meant a lack of parallelism of the ocular or visual axes. There are two varieties of squint. Paralytic squint is due to weakness of one or more of the extra-ocular muscles. Although a squint may be obvious with the eyes at rest (due to overaction of antagonistic muscles), it may only be present or accentuated, when the eyes are turned in the direction of action of the paralyzed muscle. Because of lack of movement of the affected eyeball in a particular direction, the two images of an external object fail to fall on identical points of the two retinae and diplopia or double vision results. In the case of concomitant squint, on the other hand, the squint is obvious at rest, equal for all positions of the eyes, movements of the squinting eye are usually unimpaired (when the fixing eye is covered), and double vision is rare.

With internal rectus paralysis, as in fourth nerve palsy, the squint is divergent (due to overaction of the antagonistic external rectus). With external rectus paralysis, as in sixth nerve palsy, the squint is convergent, the affected eyeball being turned inwards (due to overaction of the intact internal rectus muscle). A divergent squint may also be noted in myopia or coma.

**DIPLOPIA** Paresis of an ocular muscle may be so slight that ocular deviation or lack of movement may not be obvious. The patient may, however, complain of diplopia, seeing two images of each object. When an object is viewed, in the direction of movement of a paralyzed muscle, the image falls

directly on the macula (or sensitive spot) in the case of the good eye, but some distance away from the macula in the case of the squinting eye, thus resulting in binocular diplopia, with one image clear and the other indistinct

Testing for diplopia The patient sits with the head in a fixed position and with a red glass in front of the right and a green glass in front of the left eye. The observer moves a bar light or candle, at a distance of 15 feet, in the various cardinal positions of the eye (Maddox chart) in order to determine the direction of maximum diplopia and thus the extra-ocular muscle or muscles paralyzed. The direction, in which two clear images (red and green) are observed, is carefully noted, as well as the directions in which the images overlap, forming but one image.

Clinical significance of test (a) A diplopia, when present, increases with further separation of the images when the eye is turned in the direction of action of the paretic muscle. (b) In diplopia, the more peripherally seen image belongs to and signifies the eye that is paretic. For instance, when diplopia occurs on gazing upwards the elevator muscle is paretic. (c) When paresis affects the superior or inferior rectus, the vertical distance between the two images is maximal in the abducted position of the affected eye (horizontal eye movement away from the nose). When paresis affects the superior or inferior oblique muscle the vertical distance between the images is maximal in the adducted position of the eye (turned inwards towards the nose).

NYSTAGMUS Nystagmus can be defined as a disturbance of ocular posture which results in rhythmic oscillatory movements of one or both eyes in any or all fields of gaze. When the oscillations are equal in both directions, the nystagmus is of "pendular" type. More often it is "jerky", and two components can be differentiated, a quick component and a slow corrective component.

By convention the direction of the quick component is considered the direction of the nystagmus. Depending on the plane of movement, the nystagmus may be horizontal, vertical, or rotatory.

Maintenance of normal ocular posture The anatomical structures of importance in the maintenance of normal ocular posture are (a) The retina, and pathways carrying its impulses via the superior colliculus to the occipital cortex. (b) The vestibular apparatus. (c) Cerebellum. (d) The ocular muscles, nerves supplying them, and their nuclei. (e) Pathways connecting the above mentioned structures e.g. the medial longitudinal fasciculus which carries fibres connecting the ocular nerve nuclei to each other and to the vestibular nuclei.

Types of nystagmus The various aetiological types of nystagmus and their characteristics are as follows. (i) Retinal nystagmus Visual impairment leads to defective ocular fixation and causes nystagmus which is of pendular type. Miner's nystagmus (nystagmus seen in miners who work in dim light)

is of retinal origin Optokinetic nystagmus is a normal phenomenon of retinal origin (*vide infra*) (ii) Vestibular peripheral nystagmus The nystagmus is of jerky type, either horizontal or rotatory The quick component is directed away from the site of lesion The nystagmus becomes more coarse on looking in the direction of the quick component It is present with eyes open or closed It is usually associated with vertigo or tinnitus It is short lived as central compensation occurs after sometime (iii) Cerebellar nystagmus This occurs in lesions of the cerebellum and the cerebellar connections of the vestibular nerves The nystagmus is jerky, the quick component being always directed towards the periphery In unilateral lesions of cerebellum, the nystagmus becomes more prominent on looking towards the site of lesion It is longer lasting Vertigo is absent It is commonly seen in hereditary ataxias, multiple sclerosis and tumours and vascular lesions of brainstem and cerebellum (iv) Paretic nystagmus due to weakness of ocular muscles (v) Congenital or familial nystagmus, of uncertain aetiology, usually of pendular type

Optokinetic nystagmus (OKN) - When the eyes attempt to follow a succession of moving objects, nystagmus is observed, which consists of a slow phase in the direction of movement of the object and a quick corrective component in the opposite direction A typical example is the nystagmus seen in a person looking at the landscape from a moving train Clinically it can be demonstrated by rotating a drum with alternate black and white stripes (Barany drum) in front of the patient's eyes in different directions

Optokinetic nystagmus is a normal reflex phenomenon, which involves the occipital cortex as well as the frontal eye fields Cortical lesions which produce loss of OKN, usually involve the fast phase or the corrective component The lesion involves the frontal eye fields (Brodmann's area 8) unilaterally for horizontal OKN and bilaterally for vertical OKN

(4) Size of pupil. The size of the pupil depends on the balance between two antagonistic muscles the sphincter pupillae or circular muscle of the iris, which produces pupillary contraction, and the dilator pupillae or radial fibres of the iris, which produces pupillary dilatation The former is innervated by the third cranial nerve and the latter by the cervical sympathetic

Inequality of pupils (*anisocoria*) may be due to constriction (miosis) or dilatation (mydriasis) of one or other pupil Dilatation of a pupil may be due to third nerve paralysis, glaucoma, traumatic lesion of the eye, excessive sympathetic stimulation or the use of mydriatic eye drops

(5) Pupillary reactions. The reaction of the pupil to light (light reflex) depends on the integrity of a reflex arc, fibres of which originate in the retina, pass along with other fibres in the optic nerve, chiasma and tract, through the corpora quadrigemina to relay in the pretectal region with the Edinger-Westphal nuclei of both sides These nuclei supply the parasympathetic pupillary constrictor fibres which pass through the oculomotor nerve and reach the pupils after relay in the ciliary ganglia

In case of pupillary reaction to accommodation, the reflex arc is more complex and involves the optic pathways, the visual cortex, the middle frontal gyrus and finally the Edinger-Westphal nuclei

*Examination for pupillary reactions* (a) To test for *direct reaction* to light, the patient is asked to look into distance with one eye covered. A lighted torch (initially held out of the line of vision to avoid an accommodation reflex) is shone on the pupil of the uncovered eye, and the pupillary change noted. The other eye is then tested in similar fashion. The reflex may be described as *good*, *sluggish* or *absent*, depending on the rapidity and extent of contraction. (b) For *indirect or consensual light reaction*, one eye is exposed to light, whilst contraction of the pupil is looked for in the other eye. The indirect pupillary response is observed in the non-stimulated eye. (c) The *accommodation* (or *convergence*) reflex consists of a bilateral constriction of the pupils, associated with convergence of the eyes, when the patient is suddenly asked to fix his gaze on a near object. After asking the patient to look at some distant object, such as a clock or tree in the far distance, a small object such as a pencil or the examiner's finger is held a few inches from the patient's eyes in front of the bridge of the nose in order to prevent lowering of the lids obscuring the normal pupillary constriction and the patient is asked to fix his gaze on the new object. Return the eyes then to the distant object, the subsequent dilatation may be even easier to see. In lesions of the oculomotor nerve, both the light and accommodation reflexes are equally diminished or absent.

### Abnormal pupillary reactions

FAILURE OF THE PUPIL TO REACT TO LIGHT Loss of pupillary reflex to light is referred to as iridoplegia and is due to a lesion of the reflex pathway (a) in the afferent pathway, i.e. retina, optic nerve or chiasma. Here consensual pupillary reaction will be absent, but if unilateral both pupils will react when the normal side is stimulated, (b) in the efferent pathway, i.e. the parasympathetic supply from the midbrain. In this case the affected pupil does not react to light no matter which side is stimulated. Bilateral failure to react with intact vision, as a rule, signifies a midbrain lesion

FAILURE OF REACTION ON ACCOMMODATION Failure of the reaction on convergence is seen in Parkinsonism or may result from upper brain stem lesion

HOLMES-ADIE SYNDROME. This benign disorder of tonic pupils and absent tendon reflexes is seen in young women. The pupillary abnormality is unilateral in majority of cases. The affected pupil is dilated and does not react to light both direct and consensual. However if the patient is asked to fix her gaze at a near object, the pupil after some delay contracts and may actually become smaller than the normal one. If the patient now looks at a



Fig 13 2A

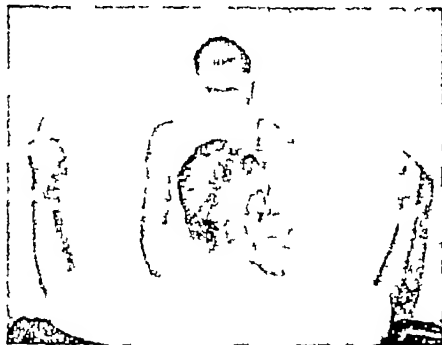


Fig 13 2B

Confrontation method of testing visual fields  
A Testing each eye separately B Testing both eyes together

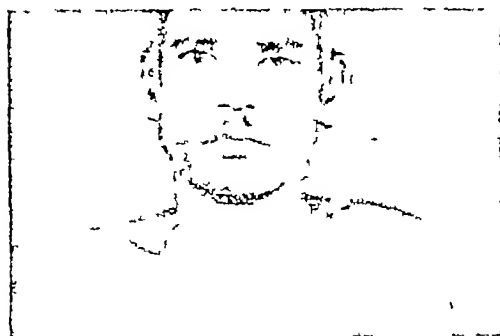


Fig 13 1 and 13 3, see pp 432 and 445 respectively

Fig 13 4, 13 5, 13 6, see overleaf

Fig 13 7 Enophthalmos in Horner's syndrome due to Pancoast's tumour (indicated by arrow)

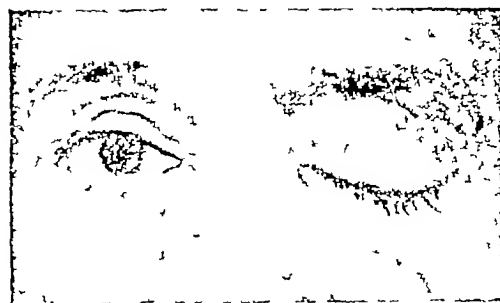


Fig 13 8A

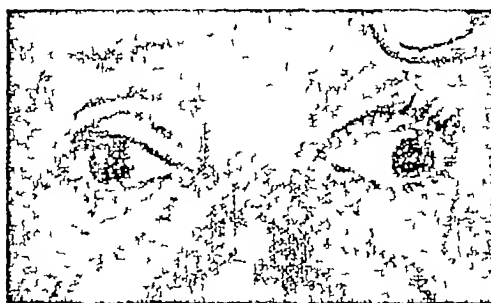


Fig 13 8B

Complete oculomotor paralysis showing  
A Ptosis B The examiner lifts up the drooping left upper lid as the patient looks upwards

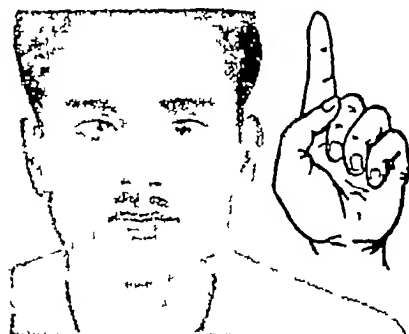


Fig 13 9 Left abducent paralysis  
The patient looks to his left, no contraction of the left external rectus muscle

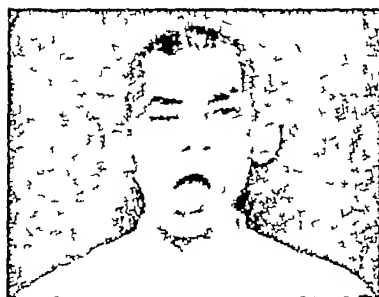


Fig 13 10 Right trigeminal paralysis When the patient opens his mouth, the jaw deviates to the right Position of upper and lower incisor

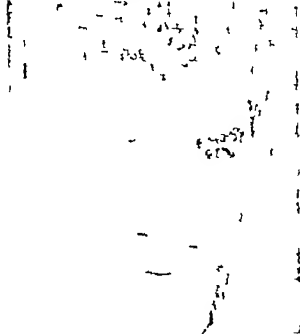


Fig 13 12A

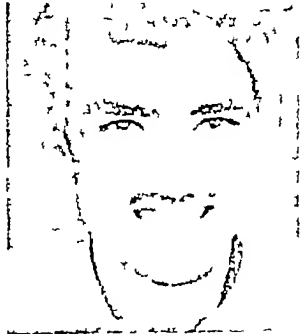


Fig 13 12B



Fig. 13 12C

facial paralysis

A The patient shows his teeth Voluntary paralysis on left side is seen B The patient begins to smile C A fully developed spontaneous smile Facial movements more pronounced on parietic (left) side



Fig 13 16 Right hypoglossal nerve paralysis Atrophy of the right half of tongue The tongue deviates to the right on protrusion



Fig 13 17 Persistent localised muscular contractions on the tongue following percussion in myotonia dystrophica

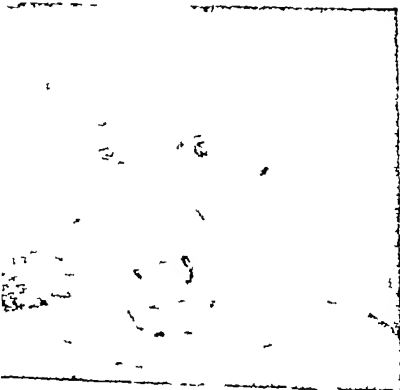


Fig. 13 18A Tongue and lips in pseudobulbar palsy



Fig. 13 18B Bilateral wasting of tongue in progressive bulbar paralysis

distant object, dilatation of the pupil proceeds even more slowly than the contraction. Some abnormality in the deep reflexes is usually present, the knee and ankle jerks being absent, occasionally there is complete areflexia.

*Wernicke's hemianopic pupil reaction* The reaction is helpful in distinguishing a tract from a radiation hemianopia. It can be demonstrated by shining a fine beam of light on to one half of the retina. In the patient with hemianopia, illuminating the blind half will produce no reaction if the lesion is situated in the early part of the optic tract, i.e. before the fibres involved in the light reflex have entered the superior collicular region.

*The Hutchinsonian pupil* When there is a rapid rise in intracranial pressure as for instance after middle meningeal artery haemorrhage, the pupil on the side of lesion is at first small and then dilates widely. This is followed by the same phenomenon in the other pupil, and is due to transtentorial herniation and resultant irritation followed by paralysis of the third nerve.

**ARGYLL ROBERTSON PUPIL** A pupil, usually small, which reacts to accommodation but not to light (reflex iridoplegia with preservation of accommodative reflex) is called an Argyll Robertson pupil. This phenomenon is extremely common in tabes dorsalis and general paralysis of the insane, but may also be encountered in encephalitis lethargica, disseminated sclerosis, intracranial tumour, midbrain lesion or chronic alcoholism. It is said that in nonsyphilitic cases the pupil is not always small.

Argyll Robertson pupil is due to selective destruction of the oculomotor nucleus or ciliary ganglion, resulting in loss of the light reflex (reflex iridoplegia). The lesion may be at more than one site — (a) Near the cerebral aqueduct in the upper half of the midbrain or (b) in the ciliary ganglion since the fibres concerned in the reaction of accommodation reaching the ciliary body bypass the ganglion. The opposite of the Argyll Robertson phenomenon (viz., loss of accommodative reflex with preservation of light reflex) is occasionally encountered in encephalitis lethargica, meningitis, syphilis, diphtheria or diabetes mellitus. It is due to selective destruction of the oculomotor nucleus, resulting in accommodative iridoplegia.

**OPHTHALMOPLEGIA INTERNA** Loss of all reflex movements of the pupils (viz., to light, accommodation, psychic and sensory stimuli), with the extrinsic muscles of the eyeball unaffected, may be encountered in neuro-syphilis and rarely after trauma or diphtheria.

**HIPPUS** Alternate dilatation and contraction of the pupil on exposure to light is a phenomenon occasionally observed in chorea, disseminated sclerosis, brain tumour, alcoholism, meningitis, hysteria or optic nerve lesion, and rarely in normal subjects. In Cheyne-Stokes respiration, the pupil may display cyclic contraction during phases of apnoea and dilatation during hyperpnoea.

**PARADOXICAL LIGHT REFLEX** Dilatation of the pupil (instead of contraction) on exposure to light may be observed in rare cases of tabes dorsalis or other neurological lesions.



## TRIGEMINAL NERVE

**Anatomy** The trigeminal or fifth cranial nerve emerges from the side of the pons in the form of two closely approximated roots, a larger sensory and a smaller motor root. The sensory fibres originate in the cells of the Gasserian ganglion and terminate in the brain stem. Whilst thermal and pain fibres terminate in the descending bulbospinal root below the pons, the fibres for touch end within a large pontine nucleus. The motor fibres of the fifth nerve originate in a small nucleus, medial to the large sensory nucleus, as well as in scattered neurones around the aqueduct of Sylvius. The nerve roots pass through the dura overlying the apex of the petrous bone. The sensory root here expands to form the Gasserian ganglion, which gives rise to three large nerve trunks, the ophthalmic, maxillary and mandibular divisions. The motor root passes forwards, beneath the ganglion, and fuses with the mandibular division. The three divisions leave via the superior orbital fissure, foramen rotundum and foramen ovale respectively.

Of the three sensory divisions, the first or ophthalmic supplies the ocular conjunctiva, lachrymal gland, inner part of the skin of the nose, scalp, forehead and upper eyelid, the second or maxillary supplies the lower eyelid, cheek, temple, upper lip, side of the nose, nasal mucosa, pharynx, soft palate and tonsils, the third or mandibular supplies the lower part of the face, lower lip, tongue, lower teeth, ear and salivary glands.

The motor root joins the mandibular division to supply the muscles of mastication (masseters, temporalis and pterygoids), the tensor tympani and (perhaps) tensor palati muscles.

**Tests of function** (1) **SENSORY DIVISIONS** These may be tested by investigating (a) various sensations over areas of skin and mucous membrane of face, corresponding to the three sensory divisions (as described above), (b) trophic skin lesions of the face (if any), (c) secretions of the salivary, buccal, and lachrymal glands. Of these signs, the first is the most reliable and consistent. In sensory involvement of the face, the patient, when drinking from a glass, may complain of the glass being broken.

(2) **MOTOR ROOT** (a) *Contraction of masseters and temporalis* The patient is asked to clench his teeth or bite hard, at the same time, contraction of these muscles is palpated over the temples and the cheeks. Asymmetrical contraction on the two sides, from paresis or paralysis, can be easily appreciated and is suggestive of motor root involvement. (b) *Deviation of jaw* The patient is asked to open his mouth wide and any deviation of the jaw noted. Slight deviation can be detected by using the relative positions of the upper and lower central incisors as guide, or alternatively a pencil or ruler is placed on the nose and aligned with the middle of the chin. The jaw deviates towards the paralyzed side (Fig 13 10) because of deficient action of the pterygoid muscle which normally tends to pull the jaw towards the midline. (c) *Side to side jaw movement* The patient is asked to move his lower jaw from side to side, preferably against resistance. There is diminished or absent movement of the jaw to the side opposite the paralysis. (d) *Hollowing above and below the zygoma*, due to wasting of the temporalis and masseters muscles, may be observed. (e) In *paralysis of the tensor tympani*, there may be partial deafness for low-pitched sounds, and in paralysis of the *tensor palati*, a slight descent of the palatopharyngeal arch. (f) In *bilateral paralysis of*

*the muscles of mastication*, the lower jaw drops and active jaw movements cannot be performed

(3) JAW JERK (see p 516)

(4) CORNEAL REFLEX (see p 512)

**TRIGEMINAL PAIN** Pain in the distribution of the trigeminal nerve is a symptom of irritation of the nerve and may be due to *trigeminal neuralgia* (*tic douloureux*), a gross lesion of the nerve, or compression of the nerve by a tumour. The latter type of pain can be distinguished from that of trigeminal neuralgia by its deep-seatedness, persistence and unremitting nature, it is also associated with sensory loss and muscle weakness in the area of its distribution

## FACIAL NERVE

**Anatomy** The facial or seventh cranial nerve, like the trigeminal, is a *mixed* nerve, with a motor and a sensory component. The motor root takes origin from a nucleus situated within the lower part of the pons. After forming a loop round the nucleus of the sixth nerve, *within the substance of the pons, the motor root of the seventh nerve leaves the ventral surface of the brain-stem*, with its sensory component, the *nervus intermedius*, and enters the facial canal or aqueduct of Fallopius through the internal auditory meatus along with the eighth nerve. Within the canal, the nerve gives off branches to the stapedius muscle, and later, the chorda tympani nerve which leaves it to join the lingual nerve. The main nerve-trunk then leaves the skull, through the stylomastoid foramen, and traverses through the parotid gland, dividing into a number of branches, which supply all the muscles of the face (except the levator palpebrae superioris), and the scalp and the platysma muscle. Between the pons and the internal auditory meatus, the nerve is in close contact with the eighth cranial nerve. The chorda tympani carries sensory taste fibres from the anterior two-thirds of the tongue. Some of the branches of the nervus intermedius supply the cutaneous area of the concha of the auricle and an area behind the ear.

**Tests of function** (1) **FACIAL EXPRESSION** The affected side of the face is immobile, there is drooping of the corner of the mouth, the nasolabial fold is obliterated and the palpebral fissure widened. Since winking no longer occurs and the lower lid is hypotonic, tears collect and tend to overflow.

(2) **SPECIAL TESTS** Weakness of different muscle groups can be tested by asking the patient to perform the following acts: (a) raise the eye-brows, so as to wrinkle the forehead, (b) frown, (c) shut both eyes tightly. The examiner then tries to open the eye against the patient's resistance. In peripheral facial weakness or palsy, the eye on the affected side remains open (*lagophthalmos*), in spite of the patient's efforts to close it, whilst the eyeball rolls upwards and slightly outwards (*Bell's phenomenon*). The presence of Bell's phenomenon indicates the intactness of the supranuclear connections. (d) Shut each eye separately. Inability to shut one eye singly may be the only sequel of an old facial palsy. It may also represent the sole defect of the upper facial musculature, in case of pyramidal or supranuclear facial paralysis. The degree to which the eyelashes are buried on eye closure should be noted and the ease with which the eyelids can be lifted. (e) Show the teeth. (f) Whistle. (g) Blow both cheeks out, fully. The cheek on the affected side puffs out with expiration, because of buccinator paralysis. (h) Smile. (i) Contract the platysma muscle. The platysma can be seen to contract, when the

patient makes a maximal effort to evert the lower lip at the same time rendering tense the skin over the anterior surface of the neck. (j) Pronounce labials. The patient may be unable to pronounce labials, such as "p", "b", "m", because of involvement of the orbicularis oris muscle. (k) Hyperacusis. Increased acuity of hearing with undue sensitivity to low tones may arise as the result of involvement of the stapedius muscle.

In unco-operative or stuporous patients, the lower facial musculature can be tested by observing the "wincing reaction", provoked by pressing firmly over the styloid process, just posterior to the angle of the jaw. In infants, facial movements can be observed during acts of crying.

**EYELID RETRACTION SIGN.** This is of value when in doubt about involvement of the facial nerve. The examiner attempts to open the closed eye forcibly with the ball of the thumb by exerting slight upward pressure on the upper lid. A positive sign consists of a reduction in amplitude and rate of vibration of the lid.

### (3) SENSE OF TASTE

**Anatomy.** Taste fibres from the anterior two-thirds of the tongue are carried in the lingual nerve to the chorda tympani nerve, and thence, through the facial nerve, to the geniculate ganglion. From there they enter the pons, either through the nervus intermedius or through the small superficial petrosal nerve, otic ganglion and third division of the trigeminal nerve. The taste fibres from the posterior one third of the tongue, pharynx and lower border of the soft palate are carried by the glossopharyngeal nerve. All these sets of taste fibres, after entering the pons, pass into the tractus solitarius, the upper part of which receives fibres from trigeminal nerve, the middle part from the pars intermedia and the lower part from glossopharyngeal nerve. From here relay neurons arise, cross the midline and proceed upwards in the tegmentum of the pons and medulla to form the gustatory fillet on the outer side of the posterior longitudinal bundle. The fillet ascends to the optic thalamus and is relayed to the hippocampus major which forms the cortical centre for taste. Lesions of the ventral posteromedial nucleus of the thalamus and the anterior portion of the second sensorimotor cortex near the insula produce impairment of taste sensation on the opposite side of the tongue.

**Method of testing.** The sense of taste is usually tested with four stock substances: (a) sugar or saccharine (sweet), (b) vinegar or sour lime (sour), (c) quinine (bitter), and (d) table-salt (salty). The patient is asked to protrude the tongue and the examiner holds it with a piece of gauze. After the tongue is dried with gauze, a small quantity of the solution or substance is applied to the tongue, on each side separately. The patient indicates the taste perceived, by pointing to the appropriate term on a card, bearing the names of the various test substances. The mouth must be rinsed out prior to testing with each new substance.

**Localization of lesion.** Facial palsy or paralysis may be of one of three types, infranuclear, nuclear or supranuclear, each type having its own characteristics.

**INFRA-NUCLEAR TYPE.** Usually unilateral and involving the entire facial musculature (*Facial monoplegia*).

(a) *Extracranial* (i) Distal to the branching of chorda tympani nerve Paralysis of facial muscles alone (ii) In the facial canal, between branching off of chorda tympani nerve and nerve to stapedius Facial paralysis and loss of sense of taste in anterior two-thirds of tongue (iii) In the facial canal, between branching off of nerve to stapedius and ganglion of facial nerve Facial paralysis, loss of sense of taste in anterior two-thirds of tongue, impairment of salivary secretion, and hyperacusis (iv) Between the internal auditory meatus and geniculate ganglion of facial nerve Facial paralysis, no impairment of taste, impairment of salivary secretion, and (frequently) nerve deafness due to associated involvement of auditory nerve In case of lack of involvement of the auditory nerve, there is hyperacusis and (perhaps) loss of affective and reflex tear secretion, with affection of the geniculate ganglion

(b) *Intracranial* Facial paralysis, unilateral or bilateral (*facial diplegia*), no impairment of taste, impaired salivary secretion, nerve deafness from

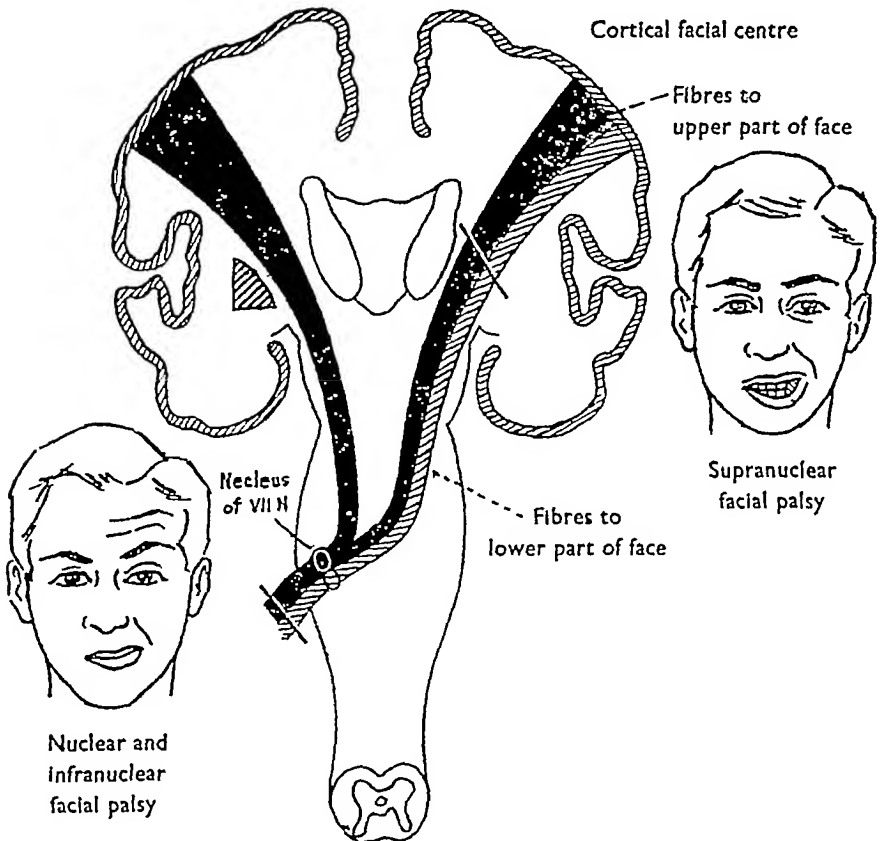


Fig 13 11 Diagram to show the bilateral cortical innervation of the upper part of the face The lower part of the face derives its supranuclear fibres from the cortex of the opposite side only (Adapted from Bing's *Local Diagnosis in Neurological Diseases*, by Haymaker)

eighth nerve involvement, multiple cranial nerve palsies, evidence of increased intracranial tension

**NUCLEAR** Upper part of face spared in majority of cases Abducent nerve paralysis in most cases Hemi-anaesthesia on the opposite side (at times)

**SUPRANUCLEAR** Practically always unilateral Minimal involvement of upper part of face because of bilateral cortical innervation of upper facial muscles (Fig 13 11) In some cases of supranuclear facial paralysis the upper face may be as severely involved as the lower This depends upon the degree of innervation from the ipsilateral hemisphere Voluntary movements of face paretic, but emotional movements (such as laughing and crying), intact or exaggerated (Fig 13 12), because of emotional movements being not dependent on the same cortical innervation as willed movements

(a) Lesion in region of cerebrum, cerebral peduncle or upper pons Facial palsy, with paralysis of ipsilateral limbs, because of association of central facial neurones with pyramidal tract

(b) Lesion in region of lower pons (pre-nuclear lesion) Facial paralysis with crossed paralysis of the limbs

*Mimic facial palsy* The upper motor neurones responsible for the emotional movements of the face seem to have a path which is separate from that of the cortico-spinal tract When there is damage to this pathway (or in lesions in the neighbourhood of the optic thalamus or involving the anterior part of the frontal lobe), emotional movements are diminished or lost while voluntary movements are retained—mimic paralysis

*Bilateral facial paralysis* (Fig 13 13) is uncommon It may be due to any of the conditions which cause unilateral palsy The causes include (1) Acute infective polyneuritis (2) Uveoparotid polyneuritis, a manifestation of sarcoidosis (3) Leprosy (4) Post-diphtheritic paralysis (5) Basal meningitis due to any cause, e.g. tuberculosis, syphilis, listeriosis (6) Myopathy, myasthenia gravis, and sometimes polymyositis (7) Benign form of spinal muscular atrophy (8) Congenital bilateral facial nuclear aplasia (Moebius syndrome), usually associated with affection of other pontine nerve nuclei (9) Bulbar palsy (10) Bulbar poliomyelitis (11) Bilateral UMN facial paralysis occurs in pseudobulbar palsy, motor neurone disease, double hemiplegia

## AUDITORY NERVE

**Anatomy** The auditory, acoustic or eighth cranial nerve is made up of two functionally distinct sets of fibres, one set (*cochlear nerve*) supplying the cochlea and subserving the sense of hearing, the other (*vestibular nerve*) supplying the vestibule and semicircular canals and concerned with the sense of equilibrium

The *cochlear* or primary auditory fibres arise from the organ of Corti, pass through the spiral ganglion and cochlear nerve, enter the pons and terminate in the ventral cochlear nucleus and tuberculum acusticum From the cells of the cochlear nucleus, a new set of fibres, the secondary auditory tracts ascend, undergo partial decussation in the pons, and pass upwards in the lateral fillet to the inferior corpora quadrigemina and internal geniculate

bodies From these, a new relay of fibres take origin, traverse through the internal capsule and terminate in the cortical auditory area or centre, situated in the upper half of the superior temporal convolution

The *vestibular* fibres originate in the vestibular ganglion, form the ventral root (the cochlear fibres forming the dorsal root) of the auditory nerve, enter the medulla and terminate in a group of nuclei The group consists of four nuclei — (a) superior, (b) lateral, (c) medial and (d) descending The nuclei are connected by afferent and efferent fibres with the following structures (i) The spinal cord Lateral vestibulospinal tract arises from the lateral nucleus Afferent spinovestibular tract fibres reach the descending and lateral vestibular nuclei (ii) The cerebellum The efferent pathway arises from medial and descending nuclei and connects it to flocculo nodular part of cerebellum The cerebello vestibular pathway arises from vermis (iii) The reticular formation (iv) Ocular motor nuclei through the fibres of medial longitudinal fasciculus (v) Cortex The cortical vestibular centre is thought to be either in the opposite temporal lobe or near the face regions of the first somato-sensory area

### Test of function

**Cochlear nerve—Acuity of hearing.** *Whispered or spoken voice* The distance at which spoken or whispered voice is heard is determined, the patient being asked to repeat words spoken by the examiner Each ear is tested separately A watch may be used for better evaluation The watch, at first held outside the range of hearing of one ear, the other ear being closed, is brought nearer to the ear until the patient is able to hear its tick The distance being noted, the examiner compares the patient's acuity of hearing with his own

**TUNING FORK TESTS** The acuity of hearing through bone is determined by placing a vibrating tuning fork (usually 256 cps) on the mastoid process and establishing the length of time that the sound is appreciated The examiner may compare the value with his own or that of a normal individual In partial nerve deafness, there is a decrease in the length of time the tuning fork is heard through bone, whilst in complete nerve deafness no bone-conducted sound is heard This test is of value only when the disturbed acuity of hearing is bilateral

**Rinne's test** This test is useful for comparing *bone* and *air* conduction of sound In the *normal* ear, air conduction is better than bone conduction ( $AC > BC$ ) A vibrating tuning fork is placed over the mastoid process on one side, when the patient ceases to hear the sound, the fork is removed from the mastoid and held before the external auditory meatus An individual with normal hearing perceives the sound anew and hears it for about twice as long as bone conduction The test is then said to be positive When sound conduction is impaired, as in the case of middle ear disease or blocking of the external auditory canal, the perception of air conduction is decreased, but that of bone conduction unaltered The tuning fork is therefore not heard when held before the ear, and Rinne's test is said to be negative

An alternative method is to hold the fork one inch from the external ear with the tines vibrating towards the external meatus for about 5 seconds and the patient is asked if the sound

is louder 'in front' The fork is then placed on the mastoid process and the patient is asked 'or back' Normally the fork will be heard better in front of the ear rather than behind Abnormally ( $BC > AC$ ), the fork is heard better behind the ear

Weber's test This test usually gives more information about the acuity of hearing than Rinne's test. The base of a vibrating tuning fork is placed on the vertex of the skull, and the patient is asked whether it is heard equally well in both the ears or is heard louder in one or other ear In a normal individual, it is heard equally well in both ears, and is not lateralized In middle ear deafness and in blockage of the external auditory canal, the sound is better heard of lateralized on the side of diminished hearing, because of increased bone conduction (*positive Weber's test*) In unilateral nerve deafness, on the other hand, the sound is lateralized to the normal side (*negative Weber's test*) In bilateral nerve deafness, Weber's test is of no value

Deafness In case of deafness, it is important to determine whether it is middle ear (conductive or obstructive) deafness or nerve (perceptive) deafness due to disease of cochlea, eighth nerve, or lesion of the pons Conduction deafness may be due to causes within the external meatus, such as wax or polyp, or within the middle ear, such as inflammation or perforation of the tympanic membrane or Eustachian canal block Nerve deafness may be due to labyrinthitis, Menière's disease, toxic nerve deafness due to drugs such as quinine, salicylates or streptomycin, senile deafness, involvement of the cochlear nerve by neuritis or tumour, or vascular lesion of the brain-stem Some reduction in hearing and difficulty in localising sound may occur with temporal lobe lesions and paroxysmal auditory sensations may form the aura of a seizure originating in this area

Loudness recruitment This test is useful in some cases of unilateral deafness Appreciation of a sound of low intensity may be diminished in the affected ear but in the case of nerve deafness as the intensity of the sound rises, the difference between the two ears diminishes and the sound comes to be heard equally well in both the ears This is suggestive of a lesion in the sensory end organs in the cochlea but may also occur in Meniere's disease and in otosclerosis

Vestibular nerve Testing of the vestibular nerve is usually not a routine procedure, being carried out only when indicated A number of techniques, involving alterations in the current of endolymph within the semicircular canals, have been evolved with a view to testing this nerve

CALORIC TEST This test allows one to investigate the labyrinthine function of each ear The patient lies with the head flexed at  $30^\circ$ , so that the horizontal canals lie in the vertical plane Irrigation of the external auditory canal with warm or cold water will cause a current in the endolymph upwards or downwards respectively This in turn stimulates the ampullae of the canal causing nystagmus During the test 250 ml of water is allowed to flow into the external auditory meatus over a period of about 40 seconds, the temperature of the water being at first  $30^\circ \text{C}$  and later  $44^\circ \text{C}$  The effects are measured as the time interval between the application of the stimulus and the end of the

resulting nystagmus. The patient lies with his gaze in the straight-ahead position. After stopping the irrigation, the time in seconds is measured during which nystagmus on forward gaze persists. The results in each ear obtained in a normal subject are charted as shown in Fig 13 14.

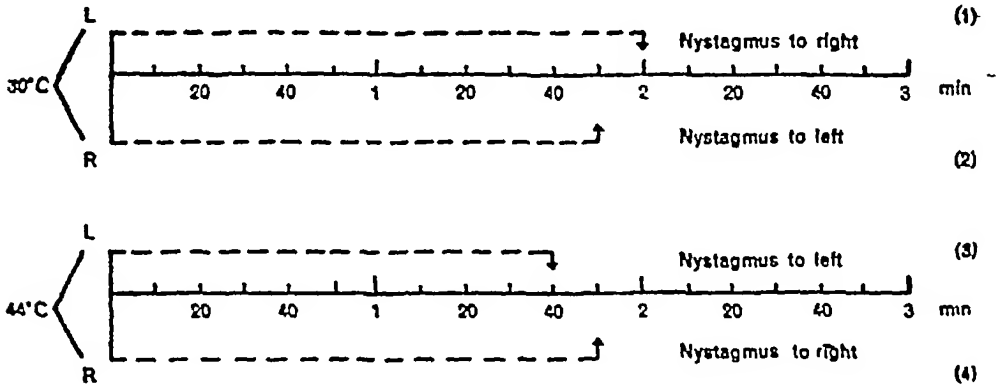


Fig 13 14 Results of caloric test to investigate labyrinthine function

*The minimal ice-water caloric test* This is a useful test and can be performed at bedside. The head is tilted forward by  $30^\circ$  and rotated to the side. One ml of icecold water is instilled and the head is rotated back to central position in 10 seconds. The response is recorded. It produces nystagmus with quick component directed to the opposite side. In an unconscious patient it also produces conjugate deviation to the left. As this response depends upon the integrity of the brainstem reflex arc, it helps to differentiate the supranuclear and internuclear ophthalmoplegias from nuclear and infranuclear types. The test can be performed on the opposite side after an interval. The test is particularly valuable in unconscious patients and must be performed in every case.

*Rotation test* Nystagmus can be induced by rotating a seated person rapidly around, about 10 times in 20 seconds, and then bringing the chair, suddenly to a stop. Normally, the quick phase of the nystagmus is towards the side opposite to the direction of the rotation. When rotation is carried out in the direction of an affected labyrinth, the response becomes more marked than when rotation is carried out in the opposite direction.

**TINNITUS** Before testing the auditory nerve, the examiner should inquire about the subjective sensation of noises in the ear. Tinnitus may be ringing, whistling, buzzing, roaring or throbbing in character, and either intermittent, paroxysmal or continuous. It is usually accompanied by deafness. Though tinnitus is a common symptom of irritative lesions of the cochlear nerve, it may be due to other causes.

*Causes of tinnitus* (1) *Peripheral causes* Impairment of function of conducting apparatus of the ear, e.g. wax in the external auditory canal, catarrh of Eustachian tube, acute labyrinthitis.

(2) *Systemic causes* Drugs like quinine, salicylates and streptomycin, fevers, hypertension, severe anaemia, aortic incompetence.



(3) *Central causes* Auditory nerve tumour, aura of epilepsy, temporal lobe lesions, brainstem lesions

## GLOSSOPHARYNGEAL AND VAGUS NERVES

The ninth and tenth cranial nerves are generally considered together since their areas of innervation overlap

**Anatomy** The *glossopharyngeal nerve* arises within the medulla oblongata from a nucleus in the floor of the fourth ventricle and emerges as a series of rootlets, somewhat lateral to the olive. Leaving the skull through the jugular foramen the nerve, after giving off meningeal and tympanic branches, proceeds to the side of the pharynx, at the level of the hyoid bone, and participates in the formation of the pharyngeal plexus. The glossopharyngeal, apart from innervating only one muscle, the stylopharyngeus is purely a sensory nerve. It also carries taste impulses from the posterior third of the tongue.

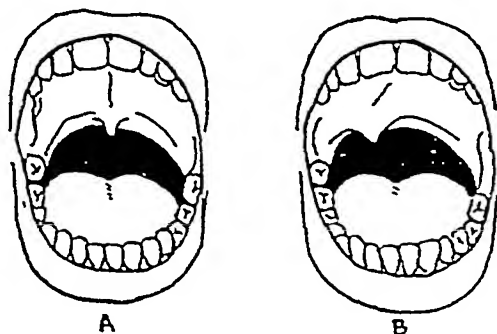
The *vagus nerve* takes origin within the medulla oblongata, from a nucleus in the floor of the fourth ventricle, leaves the skull through the jugular foramen, in company with the ninth and eleventh cranial nerves and is distributed to various structures within the neck, thorax and abdomen

The vagus or tenth cranial nerve is a *mixed* nerve with sensory and motor fibres

**Sensory fibres** Some of the sensory fibres carry impulses from the dura of the floor of the posterior fossa. An auricular branch conveys impulses from the boundary zone between the ear and the mastoid process. These fibres conveying pain terminate in the spinal tract of the trigeminal nerve. Sensory fibres concerned with the cardiorespiratory and other visceral reflexes terminate in the caudal part of the nucleus of tractus solitarius. **Motor fibres** One of the motor components arises from the nucleus ambiguus. It supplies all the muscles of the soft palate, pharynx, the cricothyroid and all the intrinsic muscles of the larynx. The other motor component arises in the dorsal nucleus of the vagus. It is motor nucleus for the smooth muscles of the trachea, bronchi, oesophagus, stomach and intestines

**Tests of function** The ninth and tenth cranial nerves are tested jointly as follows

(1) **POSITION OF UVULA**  
Normally, the uvula hangs in the midline. In case of paralysis, it deviates to the sound side. In partial paralysis, the deviation can be brought out by making the patient utter a long "a—h" (Fig 13 15)



(2) **MOVEMENT OF POSTERIOR PHARYNGEAL WALL.** Fig 13 15 Paralysis of the left 10th cranial nerve. A. Mouth opened widely. B. The patient says 'A—Ah' and the palate is pulled towards the right. When the patient utters a long "a—h" with the mouth open, a movement of the posterior pharyngeal wall towards the healthy side becomes obvious. It resembles the

“drawing aside of a curtain” It may be necessary to depress the tongue with a spatula, in order to visualize the posterior pharyngeal wall

(3) PHARYNGEAL REFLEX The pharyngeal (or “gag”) reflex is tested by stimulating the back of the throat with a tongue depressor or spatula, tickling successively both faucial pillars, or the posterior pharyngeal wall Loss of this reflex is significant of tenth cranial nerve involvement

(4) MOVEMENT OF LARYNX Whilst the patient is made to swallow some water, the force of upward movement of the larynx is assessed digitally

(5) NASAL REGURGITATION This may be seen when the patient drinks water Due to paralysis of the soft palate, the water rushes up into the nose and trickles out of the nostrils The fluid may, by going the “wrong way”, in case of paralysis of the epiglottis, bring about a cough reflex

(6) NASAL SPEECH The voice may display a “nasal twang” because of palatopharyngeal muscle palsy The speech is then described as nasal or palatal

(7) VOCAL CORDS The vocal cords may be examined with a laryngoscopic mirror, any malposition of the cords during respiration or phonation being observed The vocal cord, on the affected side, lies immobile, midway between abduction and adduction, because of the abductor and adductor muscles of the glottis being out of function

(8) SENSE OF TASTE Testing of the sense of taste over the posterior one-third of the tongue is neither as simple nor as reliable as in the case of the anterior two-thirds of the tongue, although the technique is essentially the same

**Clinical features of individual nerve paralysis** *Glossopharyngeal nerve paralysis* Isolated paralysis of the ninth cranial nerve is very rare Loss of sense of taste in the posterior one-third of the tongue, anaesthesia of the upper pharynx, loss of pharyngeal and palatal reflexes (of significance only when absent on one side), and reduction in the secretion of saliva are usually present

*Vagus nerve paralysis* In total or complete unilateral involvement or interruption of the tenth cranial nerve, at the base of the skull or intracranially, there is paralysis of the soft palate, pharynx and larynx on the ipsilateral side The soft palate on the affected side hangs flaccidly, and the voice has a “harsh twang” The vocal cord on the affected side lies immobile, in a position midway between abduction and adduction, but the voice may remain normal, in view of the compensatory shift of the opposite vocal cord across the midline There may be difficulty in swallowing, especially of solid foods, because of the interlacing of muscle fibres across the midline in the case of the pharynx Cardiac arrhythmia or tachycardia may be present With interruption of the recurrent laryngeal nerve, the voice is reduced to a whisper and is hoarse Explosive cough is no longer possible Progressive or gradually increasing disease or damage of the vagus nerve results first in paralysis of the abductors of the vocal cords, then of the tensors, and finally of the adductors

## SPINAL ACCESSORY NERVE

**Anatomy** The spinal accessory or eleventh cranial nerve is a motor nerve, which originates in part within the nucleus ambiguus of the medulla (medullary or accessory portion

and in part, within groups of cells in the anterior grey matter of the upper five cervical segments of the spinal cord (spinal portion) After emerging from the lateral surface of the cord, between the dorsal and ventral roots, the spinal fibres form a trunk, which ascends to the foramen magnum where it is joined by the accessory portion The common trunk leaves the skull, through the jugular foramen, in company with the vagus, and descends downwards in the neck to supply the sternomastoid and trapezius muscles

**Tests of function** The *trapezius muscles* are tested by making the patient shrug his shoulders, whilst palpating the muscles of both sides for evidences of contraction The procedure is then repeated against resistance, by placing both hands over the patient's shoulders In paralysis of the upper trapezius, the shoulder droops, the upper part of the scapula falls away laterally from the vertebral column, the angle between the neck and shoulder becomes more acute, and the superior angle of the scapula is displaced farther from the spine than its inferior angle

The *sternomastoid muscle*, as a rule, stands out distinctly, even at rest, and atrophy and hypotonia can be usually discovered by mere inspection To test the muscle, the patient is asked to turn his head first to the left and then to the right, each movement being opposed by placing one hand against the patient's chin, whilst palpating the contracting sternomastoid with the other In case of paralysis of the muscle, the patient is unable to turn the chin completely away from the affected side, but can turn it well towards the affected side The patient with bilateral sternomastoid weakness appears to leave the head behind on the pillow when he sits up

#### HYPOGLOSSAL NERVE

**Anatomy** The hypoglossal or twelfth cranial nerve is a purely motor nerve for the tongue. It arises from the hypoglossal nucleus in the lower third of the medulla oblongata, emerges from the ventral aspect of the medulla, and crosses the posterior fossa to leave the skull through the anterior condylar foramen Through its various branches, the nerve supplies the intrinsic muscles of the tongue (on the same side), as well as the hyoglossus, styloglossus and genioglossus muscles

**Tests of function** (1) **DEVIATION OF TONGUE** The patient is asked to open his mouth and protrude the tongue Weakness or paralysis of one side causes deviation of the tongue towards the paralyzed side, because of contraction of the genioglossus muscle (Fig 13 16) The simultaneous action of the two genioglossus muscles normally effects a straightforward pull on the tongue

(2) **LATERAL MOVEMENTS OF TONGUE** The patient is asked to move his tongue from side to side, and then to push out the cheek from within, with the aid of the tongue The strength of muscle contraction is tested, by palpation, from outside the cheek

(3) **CURLING MOVEMENT OF TONGUE** The patient is asked to curl the tongue upward and attempt to lick the nose, and then downward to lick the lower lip

(4) **ATROPHY AND FASCICULATION** of the tongue should be noted carefully At times, direct palpation of the tongue helps to confirm atrophy of one-half of the tongue

(5) **IMMOBILITY OF TONGUE** In bilateral paralysis, the tongue lies immobile on the floor of the mouth, the speech is defective and eating becomes difficult

(6) **DIMPLE ON TONGUE ON PERCUSSION** In myotonia, on tapping the surface of the tongue firmly, a clear-cut dimple or furrow appears which may persist for several seconds (Fig 13 17)

In case of facial palsy, the tongue, at times, seems to deviate to one side. In such a case, one must be guided by the relation of the tongue to the teeth, the interval between the median incisors corresponding to the midline. In trigeminal paralysis also, deviation of the jaw may cause a slight deviation of the tongue

**BULBAR PALSY** The term bulbar palsy is used to describe a clinical syndrome characterized by weakness or loss of power of muscles, which normally control the acts of swallowing, talking and movements of the lips and tongue. Since nuclei innervating these muscles are located within the medulla oblongata or bulb, the clinical condition is referred to as bulbar palsy

*Pseudobulbar palsy* The term is used when bulbar palsy results from a lesion located above the level of the medulla which interrupts the supra-nuclear tracts of lower cranial nerves (usually ninth to twelfth, fifth and seventh), resulting in an upper motor neurone type of bulbar palsy, with motor involvement of both sides of the pharynx, larynx, tongue, face and muscles of mastication (Fig 13 18 A)

The clinical manifestations are usually striking. There is dysarthria, the speech being monotonous, drawling and at times unintelligible. If palatal paresis is pronounced the voice has a nasal twang, with preponderant weakness of the lips, there is difficulty in forming "labials". The tongue which appears small and conical, can be protruded with difficulty or lies immobile. The up and down movement of the laryngeal protuberance on swallowing is weak or absent and there is dysphagia. Paroxysmal attacks of motiveless crying or laughing may occur. Unlike nuclear bulbar palsy, pseudo-bulbar palsy is not accompanied by wasting or atrophy of the affected muscles. Dysphagia while present is never as marked as in lower motor neurone bulbar palsy.

The syndrome of pseudo bulbar palsy is usually due to cerebral atherosclerosis and multiple vascular lesions in the cerebral hemispheres, affecting both pyramidal tracts above the level of brainstem. Other causes are motor neurone disease, intrinsic space occupying lesions, and degenerative diseases.

*Progressive bulbar palsy* In motor neurone disease (amyotrophic lateral sclerosis, progressive muscular atrophy), degenerative changes in the ganglion cells of the medullary motor nuclei may bring about the syndrome of a gradually developing bulbar palsy with wasting or atrophy of affected muscles. The bulbar involvement may follow, accompany or precede muscular atrophy of the hands or shoulder-girdle muscles.

The clinical characteristics are a wasted, wrinkled and shrunken tongue (Fig 13 18 B) with fibrillary twitches, inability to whistle or purse the lips, dribbling of saliva from the open mouth, slurred and often unintelligible speech with difficulty in pronouncing labials, dentals and (later) gutturals, difficult swallowing, and regurgitation of food or fluids from the nose

Other causes of lower motor neurone type of bulbar palsy are bulbar poliomyelitis, acute infective polyneuritis, botulism. When no such cause is apparent, possibility of myasthenia gravis should be kept in mind

## MOTOR SYSTEM

The motor activities of normal man comprise of reflex, automatic and purposive movements. Voluntary movement is initiated, at the highest level of cerebral function (motor cortex), by the desire or will to move. The pyramidal tract constitutes the chief outgoing efferent path, by which voluntary movement, as initiated in the cerebral cortex, is directed. Extrapyramidal and cerebellar tract systems also contribute to the co-ordinated performance of voluntary movement. Motor dysfunction can also result from involvement of lower motor neurones, impaired neuro-muscular transmission or diseases of the muscles themselves.

Because of reinforcement of pyramidal activity by the extrapyramidal pathways, total destruction of the pyramidal tract does not lead to complete paralysis. Since the centres for innervation of somatic muscles are located within the anterior horn cells of the spinal cord, destruction of these cells leads to total paralysis of the muscles they supply.

**Anatomy** Muscle power and voluntary movements depend on nervous impulses, initiated in the Betz cells (and probably a variety of other cells) within the precentral convolution of the cerebral cortex, and conducted downwards along the pyramidal or upper motor neurone fibres, within the pyramidal tract, and through the internal capsule, midbrain, pons and medulla, to terminate in the grey matter (anterior horn cells and internuncial fibres of the intermediate zone) of the spinal cord. From these lower centres in the cord, a further relay of fibres, the lower motor neurones, take over the function of conduction of nervous impulses to the various muscles of the body. Whilst the pyramidal or upper motor neurone fibres affect muscles in terms of movements, the lower motor neurone fibres supply groups of muscle fibres within individual muscles.

In the precentral convolution, various parts of the body are represented, in terms of movements of muscle groups, in reverse order, the head and neck being lower-most and the foot and toes upper-most at the vertex. The actual order of represented muscle movements is from below upwards: larynx, pharynx, palate, mandible, tongue, face, neck, thumb, fingers, forearm, arm, trunk, thigh, leg, foot and toes.

The pyramidal tract occupies the posterior one-third of the anterior limb, the genu and anterior two-thirds of the posterior limb of the internal capsule, the middle three-fifths of the basis pedunculi in the mid brain, and splits into several groups of fibres within the pons, to reunite again into a compact tract within the medulla. At the junction of the medulla and cord and below this level, the pyramidal tract undergoes partial decussation, finally to end within the grey matter of the spinal cord. Some of the fibres descend uncrossed as the anterior

UMN - pseudotumor MN, Multiple vascular lesions  
degenerative disease

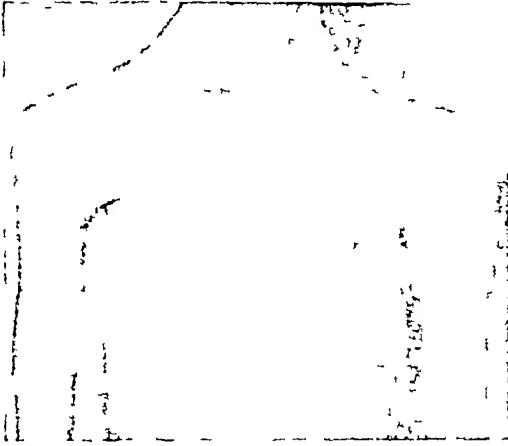


Fig 13 19 Wasting of the right deltoid muscle



Fig 13 21 Bilateral atrophy of sternomastoids in a case of dystrophia myotonica

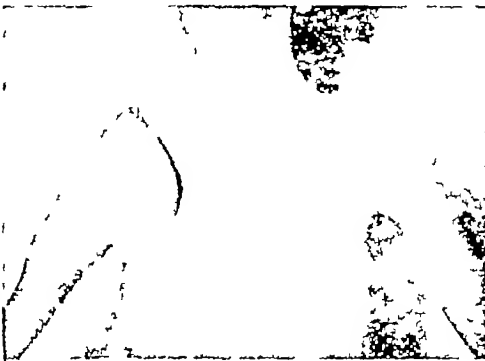


Fig 13 22 Bilateral winging of scapulae in progressive muscle wasting



Fig. 13 20 Wasting in facio-scapulo-humeral myopathy

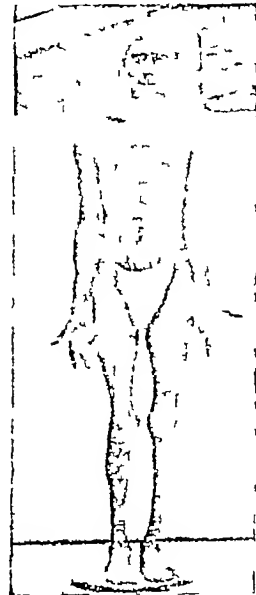


Fig 13 23 Wasting of calf muscles and lower thirds of the thigh in peroneal muscular atrophy producing "inverted champagne bottle" limb

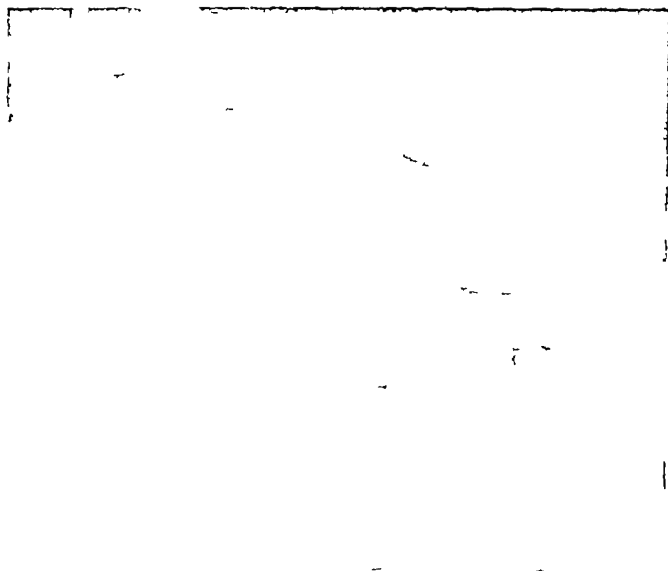


Fig 1324 Left median nerve paralysis The left index finger fails to lift. The site of injury is indicated by the arrow

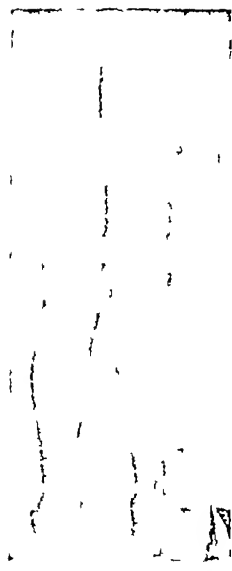


Fig. 1325 Pseudo hypertrophy of calf muscles and glutei in pseudo hypertrophic muscular dystrophy

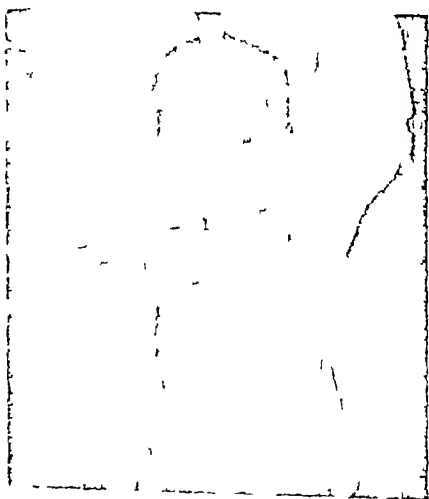


Fig 1326 Weakness of chest and shoulder muscles in pseudo hypertrophic myopathy allows the child to slip through when an attempt is made to lift him by putting the hands under the armpits



Fig 1327 Extreme degree of hypotonia in benign non progressive congenital myopathy

corticospinal tract In the brainstem, corticobulbar fibres supply the cranial nerve nuclei The facial nucleus concerned with the lower face, the fifth nerve motor nucleus, the hypoglossal and accessory nucleus receive fibres from the opposite hemisphere only The other cranial nerve nuclei are supplied by both hemispheres From the cells of the motor nuclei of the brain-stem and anterior horns of the spinal cord, a second relay of fibres, the *lower motor neurones*, take origin, pass through the anterior roots, spinal or cranial nerves, and peripheral nerves, to end in small groups of muscle-fibres within the various muscles of the body

## SCHEME OF EXAMINATION

An examination of the motor system implies an investigation of the following motor functions (1) state of nutrition, (2) muscle tone, (3) motor power, (4) muscular co-ordination and (5) involuntary muscle movements

## STATE OF NUTRITION AND WASTING

The nutritional status of muscles, whether soft, flabby and wasted, normal, increased in bulk, should be carefully assessed by inspection and palpation

The size of a voluntary muscle varies with age, sex, body build, state of nutrition and training of the individual

**Atrophy** As applied to muscles, the term atrophy implies a loss of bulk, a muscle once large becoming smaller in size (Figs 13 19, 13 20, 13 21, 13 22, 13 23, 13 24) In determining the presence of atrophy or wasting, the bulk of the muscle is determined by inspection and palpation, and comparison made with neighbouring muscles, with the same muscle on the opposite side of the body and with general muscular development Minor differences in the circumference of corresponding extremities is not a reliable means of determining atrophy, since an asymmetrical development of muscles on the two sides, as in the case of the calves, may be present Comparative measurements are of value, however, in determining the progression of atrophy

Muscle atrophy is usually due to a lesion of anterior horn cells, nerve root, peripheral nerve or muscle itself It has also been reported with lesions of parietal lobe Disuse atrophy may occur in long standing upper motor neurone lesions, prolonged immobilisation of a joint as in plaster casts following fractures, or diseases of joints such as tuberculosis and rheumatoid disease

Atrophy resulting from disease of the lower motor neurone or nerve is almost always associated with significant weakness of the muscle On the other hand, when atrophy results from a debilitating systemic disease or disease of muscle, the vigour of contraction remains fairly good

**Hypertrophy.** An increase in bulk or size of muscle is described as hypertrophy It should be remembered that a muscle of normal size may appear hypertrophic in the midst of atrophic neighbouring muscles Hypertrophy



of muscles may be observed with muscular dystrophies and polymyositis. It is usually best observed in the calf muscles and deltoid. The muscles should be palpated in order to determine their consistency.

It is rather unfortunate that terms such as "hypertrophy" and "pseudohypertrophy" are used to describe the neuromuscular disorders. Usually the term hypertrophy is used when the enlargement is associated with normal power and pseudohypertrophy when it is associated with weakness. Neuropathological studies have shown that at least in the early stages of muscle dystrophy the enlargement of muscle is associated with true increase in the diameter of muscle fibres and thus till the later stages is indeed responsible for the enlargement. In the later stages there is replacement of muscle fibres by fat and connective tissue and these are then responsible for the enlarged appearance. Muscle enlargement is a prominent feature of myotonia congenita, hypertrophy musculorum vera (the exact clinical status of this disorder is not known), gigantism and acromegaly. It is also a prominent feature in the X-linked muscular dystrophies, the calves being particularly involved (Fig. 17.25), (the calf muscle is often of normal power till late in the disease), other muscles involved being the quadriceps, deltoids and triceps. Carriers of X-linked dystrophy often show muscle enlargement (especially calf). It is less common in other forms of dystrophy. Muscle enlargement has been well documented in benign forms of spinal muscular atrophy, limb girdle muscular dystrophy and rarely in polymyositis.

Loose shoulders. In progressive muscular dystrophy the child if lifted with the examiner's hands in the axillae, will tend to slip through the examiner's hands (Fig. 13.26).

## MUSCLE TONE

The mild degree of tension or state of continuous muscle contraction, found in normal healthy muscle, is referred to as muscle tone or tonus. The tone of a muscle varies with the needs of the moment and is governed reflexly by a variety of nerve impulses from various nerve centres in the brain and spinal cord, special end organs, skin and muscles. These impulses exert their influence on two sets of opposing mechanisms, the one concerned with excitation and the other with the inhibition of tone. Normally, these two influences are well-balanced, resulting in normal muscle tone. The efferent impulses from these systems reach the anterior horn cells and their cranial homologues, via the pyramidal and extrapyramidal tracts, and influence the muscles through low frequency asynchronous discharges.

Methods of investigation. Examination of muscle tone is carried out as follows:

(1) RESISTANCE TO PASSIVE MOVEMENT. The patient is asked to relax completely as far as possible, since voluntary relaxation of the part examined is most important. The patient's attention may be diverted during the test.

for this purpose. The muscle tone is then assessed in a systematic manner, at the joints of the upper and lower extremities, by passive movements. The resistance offered by the muscles during passive handling or movement represents the degree of tone. The resistance to handling of the limb may be normal, increased (hypertonia), decreased (hypotonia) or even absent (atonia). The range of movement at a given joint tends to be increased in hypotonia.

Both upper and lower extremities are tested in turn by being moved passively, at the various joints, through flexion, extension and rotation. At each joint, the degree or type of resistance to movement and range of movement are observed closely.

(2) **INSPECTION AND PALPATION** Observation and palpation of superficial muscles may give a rough idea of the state of muscle tone. Whilst in hypertonus, the muscle bellies stand out prominently and display increased convexity, in hypotonus, there is obliteration of contour of superficial muscle bellies. Direct palpation of a muscle gives an idea of muscle tone at rest and is particularly useful in determining increase or loss of tone (not detectable during passive movement of a joint), the muscle being either firm to the feel and resistant to lateral movement, or soft to palpation and easily displaceable.

(3) **PENDULOUSNESS** On shaking or swinging a normal limb or extremity passively, there is a regular pendular movement which diminishes steadily and in an even manner. For testing pendulousness in the upper extremity, the arm of the patient is briskly flipped away from the body, the speed, range and regularity of movement of the arm swing being closely observed. In the case of the lower extremity, the patient is made to sit on the edge of a table or bed with the leg hanging down, the leg is then suddenly jerked upwards and dropped and the pendular movements watched. Each limb should be tested individually, the range and type of movement being compared on the two sides.

(4) **ABNORMAL POSTURES** Whilst in hypertonia an extremity tends to maintain a fixed posture of over-extension or over-flexion, in hypotonia the posture of the limb is usually dictated by the factor of gravity.

In pyramidal rigidity or hypertonia, the posture of the body and limbs depends on the site, nature and degree of lesion. In cerebral apoplexy, with spastic hemiplegia, the trunk is extended, the arm adducted at the shoulder and flexed at the elbow and wrist, and the leg extended at all joints. In spinal paralysis, the lower extremities may be in a state of extension or flexion (paraplegia in extension or flexion). In hypertonia of extrapyramidal origin, as in Parkinsonism, the entire body is in a state of flexion, the posture being diagnostic of the disease. In hypotonia, the upper limb shows extension at the elbow, pronation and flexion at the wrist, and hyper-extension of the metacarpo-phalangeal joints.

(5) **POSTURAL FIXATION** Normally, the desired posture of a limb can be maintained for some length of time without voluntary correction. With alteration of muscle tone, postural fixation of a limb may become impaired. It is usually tested by asking the patient to shut his eyes and extend the arms horizontally in front. A hypotonic arm tends to drop or deviate downwards. On sharply tapping the out-stretched hand, a hypotonic limb tends to rebound or overshoot its original position and oscillates several times before regaining its original posture. A normal arm returns promptly to its original position without oscillating.

**Abnormalities of muscle tone** **HYPERTONIA** (Hypertonus, rigidity, spasticity) Increase of tone may be of several types

*Spasticity* This is a variety of increased tone, where, as the joint is passively flexed or extended, there is increased resistance to begin with, but as the movement is continued, the resistance disappears suddenly ("clasp-knife" type). As a rule, this type of spasticity ("moulding" spasticity), which is typical of a *pyramidal* lesion, is more marked in one direction than in the other (being more marked in the flexors of the arm and the extensors of the leg), and more marked during a rapid than a slow movement.

*Rigidity* The resistance to passive movement in this type of hypertonia is equally marked in both agonists and antagonists (*plastic* or *lead pipe* type of rigidity). This type of rigidity, is a characteristic feature of extra-pyramidal system disease.

*Cog-wheel rigidity* In this type the agonists and antagonists contract alternately and regularly during the movement. It is found in extra-pyramidal disease and often when there is associated tremor. The phenomenon can be demonstrated well if the examiner grips the fingers of the patient and moves the hand to and fro at the wrist joint. It is markedly increased if the patient is made to clench the opposite fist while the test is performed. The limb, after being moved from its original attitude, keeps the new position with the same degree of resistance to movement as in the original (*plasticity*).

*Hysterical rigidity* In this type, the resistance to passive movement is roughly proportional to the effort made by the examiner to move the limb. It is typical of hysteria.

*Myotonia* This is a state in which there is a prolonged after-contraction of the affected muscles which persists after the voluntary movement has ceased. It is best seen in the flexors of the fingers so that the patient has difficulty in relaxing his grip.

*Decorticate and decerebrate rigidity* Observation of the position of the body in an unconscious patient may help to localise the lesion within the central nervous system. Generally disturbances of tone and posture result from lesions which produce decortication, i.e. separation of the motor cortex from



Fig 13.28 Hyponotia in amyotonia congenita

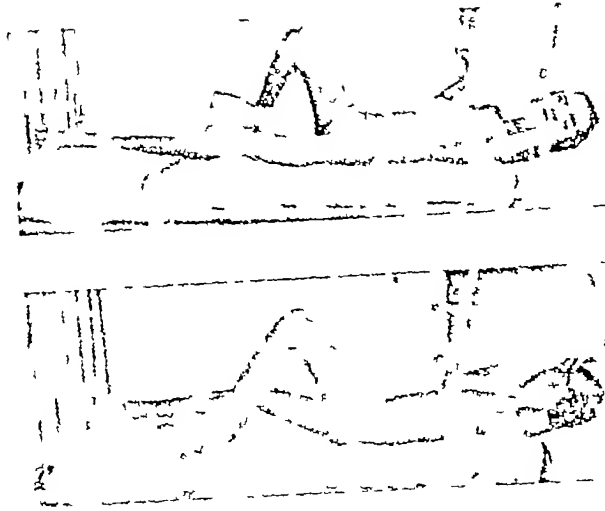


Fig 13.30 Brudzinski's neck sign

Fig 13.29, see p 476

Fig 13.31, see p 476



Fig 13.32A Trapezius The patient is shrugging his shoulders against resistance

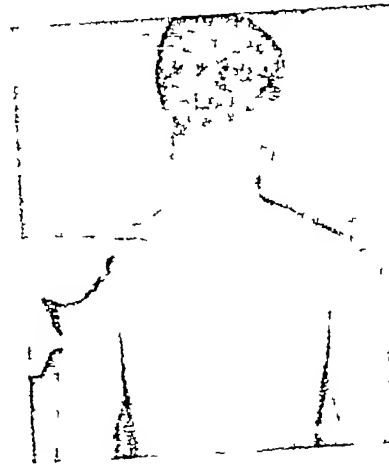


Fig 13.32B Trapezius patient is bracing the shoulders backward.



the spinal cord, or decerebration in which the cerebrum is separated from the brain stem either functionally or anatomically. A painful stimulus is often required to elicit the responses.

In decorticate position the posture is that of hemiplegia with flexion of the upper limbs and extension of the lower limbs.

In decerebrate rigidity there is extension of all limbs with internal rotation of the arms and plantar flexion of the feet.

**Gegenhalten** This refers to an increase in resistance in response to passive movements. The patient actively contracts the muscle concerned and prevents its passive extension by the examiner. This may be observed in diffuse frontal lobe disease and lesions of the basal ganglia.

**Scarf sign** In lower motor neurone disease with marked hypotonia, it may be possible to pull each forearm towards the opposite shoulder and almost wrap them around the neck like a scarf.

**HYPOTONIA** (Hypotonous, flaccidity) A diminution of muscle tone is characterized by a decreased resistance of the limb to passive movement as well as an increased range of movement. It is observed in lower motor neurone lesions, in cerebellar diseases, in amyotonia congenita, myopathy, in upper motor neurone lesions during the stage of "neuronal shock", and in some extra-pyramidal disorders like "limp" chorea (Fig 13 27, 13 28).

**Tests for rigidity in meningitis** The following signs are of diagnostic value in suspected cases of meningitis or meningeal irritation (meningism).

**NECK RIGIDITY** Resistance to passive flexion of the head is an early sign of meningitis. Resistance due to spasm of the extensor muscles of the neck prevents effective flexion, when the examiner after putting his hand underneath the patient's occiput tries to approximate the chin to the chest.

**KERNIG'S SIGN** The patient's hip being flexed, the examiner attempts to extend the knee as far as possible without producing appreciable pain. Normally, the knee can be extended so that the angle between the posterior surface of the thigh and leg is about  $135^\circ$  or  $1\frac{1}{2}$  right angles. With a positive Kernig's sign (Fig 13 29), extension of the knee is limited by spasm of the hamstring-muscles and pain arises as the result of the stretching of the spinal nerve-roots of the lower limb, the lower end of the subarachnoid space of the spinal cord being distended and spinal meninges inflamed.

**BRUDZINSKI'S SIGNS** These signs are also found in meningitis, but much less frequently than Kernig's sign. In Brudzinski's *neck sign*, there is flexion of the hips and knees on flexing the neck or turning it to one side (Fig 13 30), whilst in the *leg sign*, on flexing one lower extremity, the opposite limb flexes automatically.

In young children too much significance should not be attached to the absence of Kernig's or Brudzinski's sign.

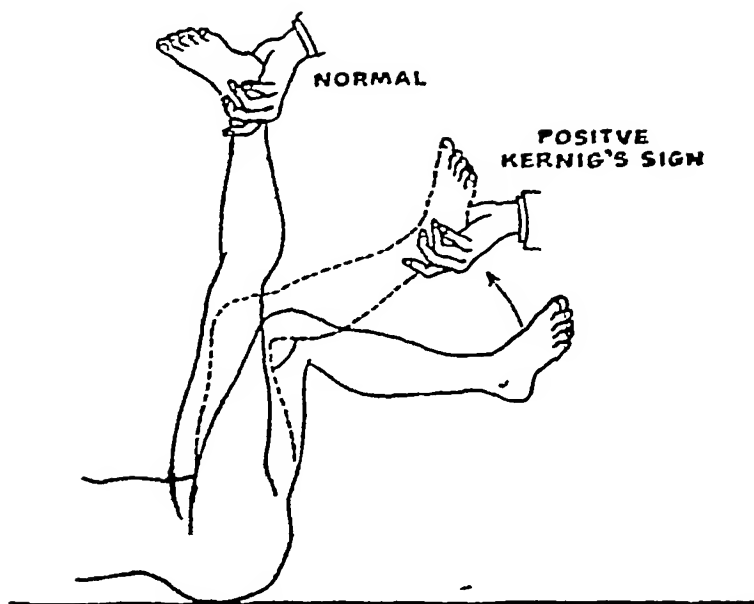


Fig. 13.29 Kernig's sign

**STRAIGHT LEG RAISING TEST (S L R , Lasegue's sign)** The test is carried out with the patient lying on his back and both legs fully extended. Each leg is passively flexed at the hip while the knee joint is kept extended (Fig. 13.31)

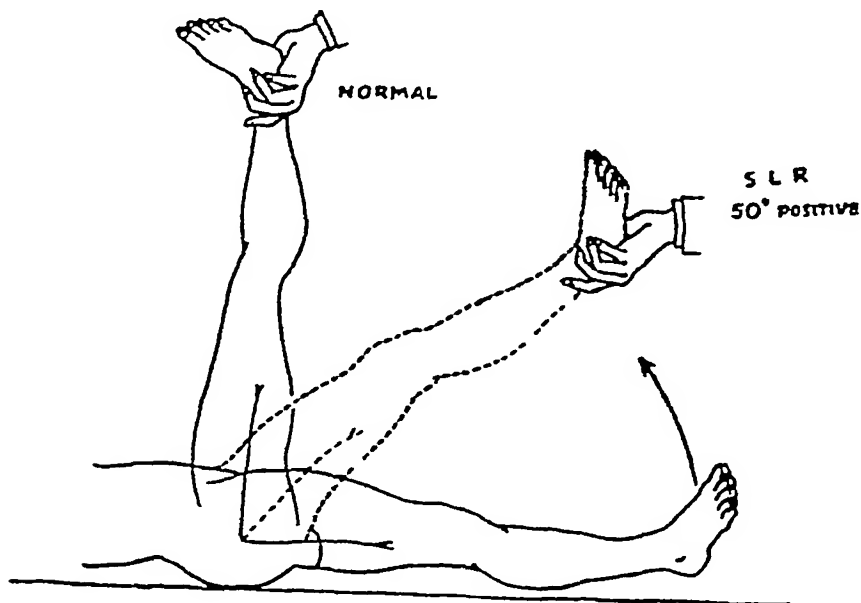


Fig. 13.31 Lasegue's sign

Normally, the straight leg can be lifted to an angle of  $65^{\circ}$  to  $90^{\circ}$  with the horizontal, the distance depending on the tightness of the hamstring muscles. Lasegue's sign is said to be positive when limitation of movement and pain are present before the leg can be elevated to the normal extent. The result of the test can be indicated by denoting the angle at which pain is felt. Thus S L R  $+ 50^{\circ}$  means that pain is first elicited when the extended leg is being flexed at the hip joint to the extent of about  $50^{\circ}$ .

In acute low back pain and sciatica, S L R may be  $5^{\circ}$  to  $10^{\circ}$  positive, with rest and treatment the angle gradually increases. Contracture of reflex origin and pain of radicular origin are responsible for a positive Lasegue's sign.

When the leg is flexed to the extent when pain begins, dorsiflexion of the foot increases the pull on the sciatic nerve without altering the tension of the hamstrings and this increases sciatic pain but not pain from other causes.

*Reversed* Lasegue's sign refers to pain with hyperextension of the hip joint in high lumbar disc lesions. Eventually in such a case, a *crossed* Lasegue sign i.e. pain down the back of one leg when the opposite leg is passively elevated is elicited.

Kernig's sign supplies the same information as S L R. However, since it is a classical sign for the diagnosis of meningitis, it continues to be mentioned and used.

**TRIPOD SIGN** Another method of demonstrating resistance to spinal flexion is to ask the patient to carry out active movements which normally involve flexion of the spine such as sitting up in bed. The patient tries to sit up by supporting himself with his hands placed far behind him in the bed to take the weight off the spine and prevent its flexion. This sign is frequently seen in the early stage of poliomyelitis. The child may also be asked to approximate his chin to his bent knees, this he fails to do.

**MYOTONIC REACTIONS** In certain types of neuromuscular disorders, contraction of an involved muscle or muscles may persist for some time after its voluntary contraction has ceased. To test for myotonia, the patient is asked to clasp the examiner's hand strongly but momentarily (as in a handshake). A persistence of contraction of the flexor muscles of the fingers makes it difficult for the patient to release his grip.

**PERCUSSION MYOTONIA** A persistent localized muscle contraction after a sharp tap with a percussion hammer is referred to as percussion myotonia. A characteristic depression of the deltoid muscle, as the result of persistent muscle fibre contraction, can be induced by sharply tapping the deltoid on one or other side. A similar tap over the thenar eminence of the hand may result in a quick but prolonged muscle contraction resulting in apposition of the thumb. The sign is a characteristic feature of myotonia dystrophica.

**MYOIDEMA (Myotatic irritability)** At times a localized "hillock-like" bulging of a superficial muscle such as the pectoral muscle may arise temporarily, at the site of percussion. Such a condition is suggestive of malnutrition, general debility or myxoedema, but may occur in perfectly normal individuals.



## MOTOR (OR MUSCLE) POWER

**Tests of function** There are two main methods of investigating muscle power

(1) The patient tries to move the muscle, under investigation, against resistance offered by the examiner (active method)

(2) The muscle to be tested is put in its final position at the end of its range of action, and the patient asked to hold the muscle in that position, while an effort is made by the examiner to move it. For instance, to test the dorsiflexors of the ankle, the joint is passively dorsiflexed and the patient asked to retain this position, whilst the examiner makes an effort to plantar flex the joint. This method is frequently easier to carry out in patients unable to understand properly orders given during examination. It is easier to put a limb in a certain position and ask the patient to hold it

**GRADATION OF MUSCLE POWER.** For recording the power of contraction of each muscle, the following system of gradation is internationally used

0—No contraction

1—Flicker or trace of contraction

2—Active movement with gravity eliminated

3—Active movement against gravity

4—Active movement against gravity and moderate resistance

5—Active movement against gravity and full resistance (Normal power)

It is also advisable to feel or palpate the contraction of the muscle, whenever accessible. The nature and object of examination must be explained to the patient first in order to secure his co-operation

**Tests of function for individual muscles** For diagnosis, it is essential to have a knowledge of (1) the innervation, (2) mode of action and (3) method of testing of each individual muscle of the body

**TRAPEZIUS** (Accessory nerve, C 3, C 4)

*Action* Adduction of scapula with elevation of its vertebral border

*Tests* (1) *Upper portion* The patient is asked to elevate his shoulder against resistance (Fig 13 32A) (2) *Middle portion* The patient is asked to brace the shoulder backward (Fig 13 32B). Adduction of the arm against resistance tends to intensify the winging of the scapula.

**RHOMBOIDS** (Nerve to rhomboids, C 5)

*Action* Retraction (adduction) of scapula and deviation of its vertebral border

*Test* With hand on hip, and arm held backwards and medially, the patient attempts to brace his shoulder backward against resistance, or the examiner attempts to force the elbow laterally and forward, observing and palpating the muscle bellies medial to the scapula (Fig 13 33)

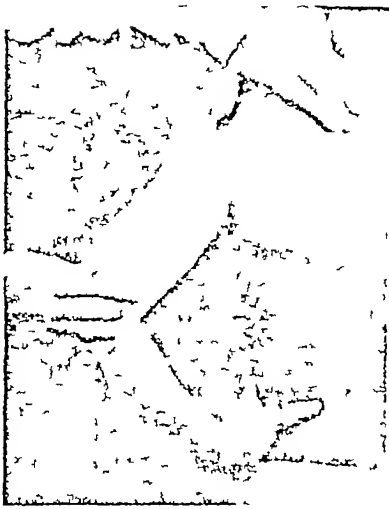


Fig 13.33 Rhomboids Hand on hip the patient tries to force his elbow backwards

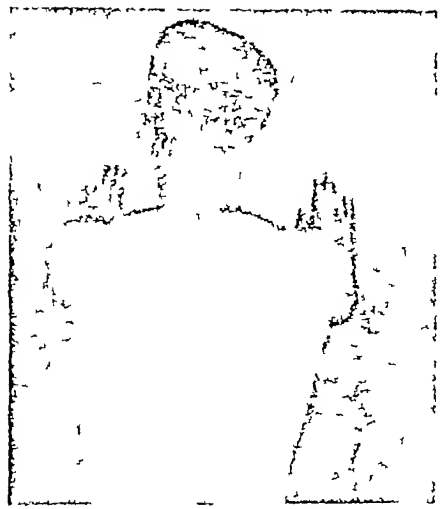


Fig 13.34 Serratus anterior The patient pushes his arms forward against some immovable object.



Fig 13.35 Supraspinatus The patient is trying to abduct the arm against resistance



Fig 13.36 Infraspinatus With the flexed elbow kept to the side the patient attempts to turn the forearm backwards against resistance



Fig 13.37A Pectoralis major, clavicular part. The patient raises the arm forward above the horizontal and is trying to adduct it against resistance

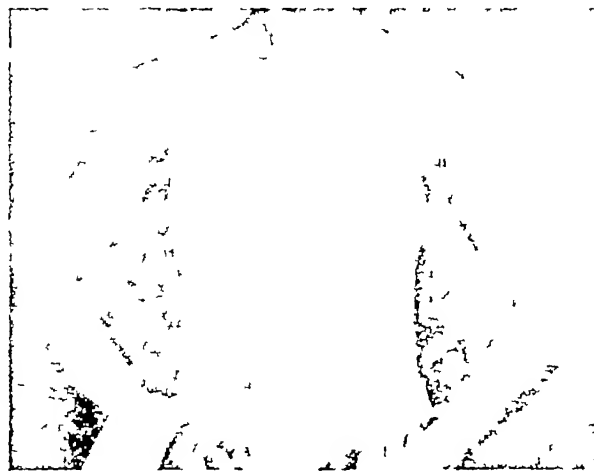


Fig. 13.37B Pectoralis major, sternal part. The patient raises the arm to below the horizontal and is trying to adduct it against resistance

Fig. 13.38A Lattismus dorsi The patient is trying to adduct the arm against resistance

Fig. 13.38B Lattismus dorsi While palpating the muscle bellies the patient is asked to cough

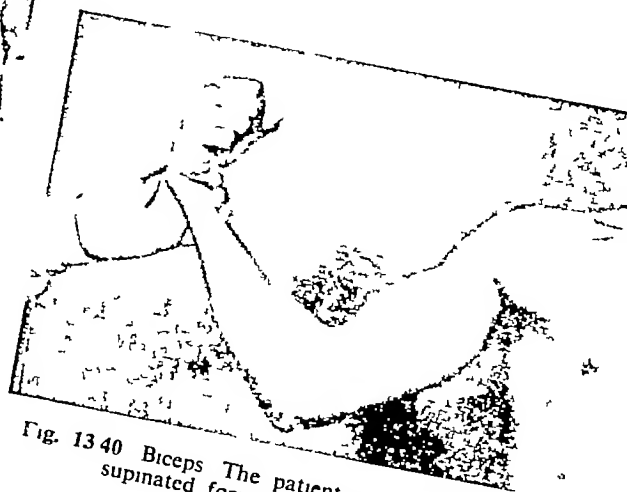


Fig. 13.40 Biceps The patient is trying to flex the supinated forearm against resistance

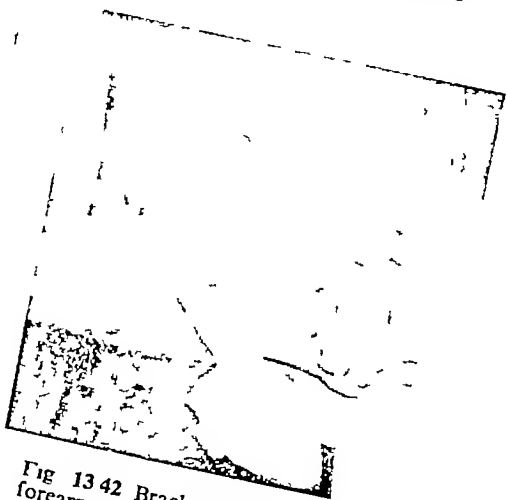


Fig. 13.42 Brachioradialis With the forearm in a pronated position the patient is trying to flex it against resistance

Fig. 13.41 Triceps The patient attempts to extend the elbow against resistance

**SERRATUS ANTERIOR** (Nerve to serratus anterior, C 5, C 6, C 7)

*Action* Lateral and forward movement of scapula, with its close approximation to the thorax Assistance in upward rotation of the scapula

*Test* Forward thrust or pressure either against the wall or against resistance offered by the examiner (Fig 13 34) In paralysis, the entire scapula, particularly its inferior angle, shifts backward and away from the thorax, producing a characteristic winging effect

**SUPRASPINATUS** (Suprascapular nerve, C 5, C 6)

*Action* Initiation of abduction of the arm from the side of the body

*Test* Abduction of the arm against resistance The muscle belly can be felt and sometimes seen (Fig 13 35)

**INFRASPINATUS** (Suprascapular nerve, C 5, C 6)

*Action* Lateral or external rotation of the arm at the shoulder joint

*Test* With the elbow kept to the side and flexed to 90°, the patient tries to carry the flexed forearm backward against resistance, or the patient resists the examiner's attempt to push the hand medially towards the abdomen (Fig 13 36)

**PECTORALIS MAJOR** (Clavicular part lateral pectoral nerve, C 5, C 6, Sternal part medial pectoral nerve, C 6, C 7, C 8, T 1)

*Action* Adduction and medial rotation of arm

*Tests* (a) Clavicular part The arm is raised forwards above the horizontal level and adduction carried out against resistance The clavicular part of the muscle can be seen and felt (Fig 13 37A) (b) Sternal part The patient raises the arm to a level below the horizontal and then tries to adduct it against resistance offered by the examiner (Fig 13 37B)

**LATTISSIMUS DORSI** (Nerve to latissimus dorsi, C 6, C 7, C 8)

*Action* Adduction, extension and medial rotation of arm

*Tests* (a) With the arm adducted to horizontal, the patient tries to carry the arm downwards and backwards, against resistance applied under the elbow The muscle can be seen and palpated in and below the posterior axillary fold (Fig 13 38A) (b) A brisk contraction of the muscle bellies can be felt at the inferior angle of the scapula on asking the patient to cough (Fig 13 38B)

**DELTOID** (Circumflex nerve, C 5, C 6)

*Action* Abduction of arm. Flexion and medial rotation of arm (anterior fibres), extension and lateral rotation of arm (posterior fibres)

*Test* The arm is held in abduction, almost horizontally An effort is made to depress the elbow whilst the patient offers resistance (Fig 13 39)

**BICEPS** (Musculo-cutaneous nerve, C 5, C 6)

*Action* Flexion and supination of forearm.  
*Test* Flexion of forearm against resistance (Fig 13 40) The forearm must be kept in a position of supination, to avoid the action of the brachioradialis

TRICEPS (Radial nerve, C 6, C 7, C 8)  
*Action* Extension of forearm  
*Test* The forearm being kept in a flexed position, the patient either tries to extend the forearm against resistance, or resists the attempt of the examiner to flex the forearm further (Fig 13 41) Gravity must be eliminated by raising the arm

BRACHIORADIALIS (Radial nerve, C 5, C 6)  
*Action* Flexion of forearm at elbow  
*Test* With the forearm midway between pronation and supination, flexion of forearm is attempted against resistance The muscle belly can be seen and felt (Fig 13 42)

EXTENSOR CARPI RADIALIS LONGUS (Radial nerve, C 6, C 7, C 8)  
*Action* Extension and radial flexion of hand at wrist  
*Test* With forearm pronated and fingers extended, patient tries to extend (dorsiflex) the wrist to the radial side, against resistance applied to the dorsum (Fig 13 43)

SUPINATOR (Radial nerve, C 5, C 6)  
*Action* Supination of forearm  
*Test* With the forearm in full extension and supination, the patient resists attempt to pronate forearm (Fig 13 44)

EXTENSOR DIGITORUM (Radial nerve, C 7, C 8)  
*Action* Extension of proximal phalanges of the fingers  
*Test* The patient is asked to resist attempts to flex the fingers at the metacarpophalangeal joints (Fig 13 45)

EXTENSOR CARPI ULNARIS (Radial nerve, C 7, C 8)  
*Action* Extension and ulnar deviation of the hand  
*Test* The patient tries to dorsiflex the wrist joint to the ulnar side against resistance (Fig 13 46)

ABDUCTOR POLLICIS LONGUS (Radial nerve, C 7, C 8)  
*Action* Abduction of the metacarpus of the thumb in the same plane at right angles to the palm  
*Test* The forearm is held midway between pronation and supination The metacarpal of the thumb is stabilized by the examiner, and the patient

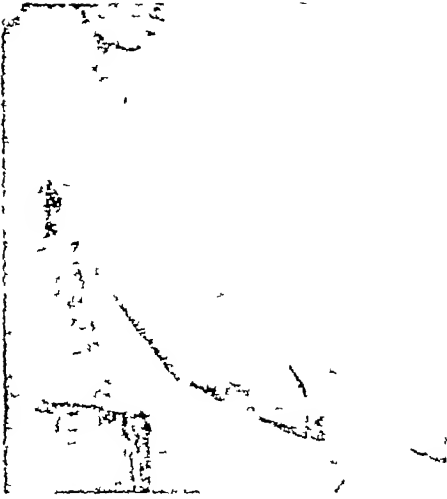


Fig 1343 Extensor carpi radialis longus With the fingers extended the patient is trying to extend the wrist to the radial side against resistance

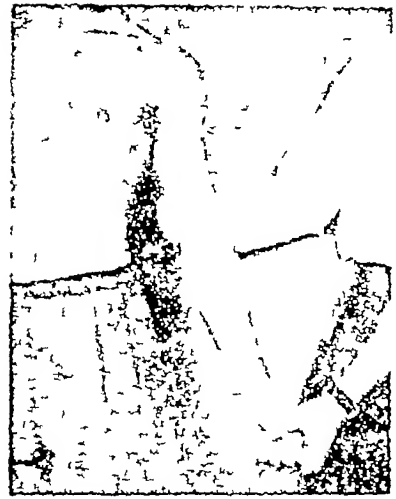


Fig 1344 The patient extends the arm by the side and resists the examiner's attempt to pronate the hand

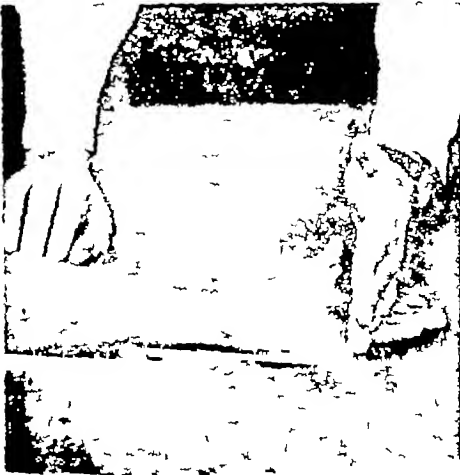


Fig 1345 Extensor digitorum The examiner attempts to flex the patient's extended fingers at the metacarpophalangeal joints

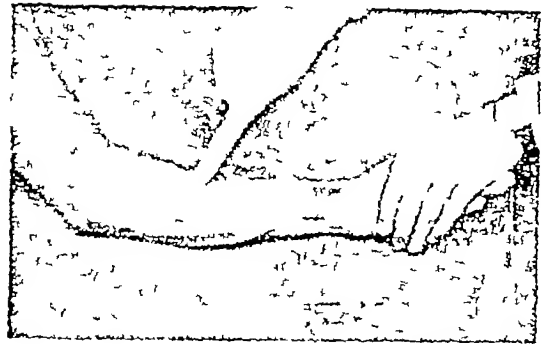


Fig 1346 Extensor carpi ulnaris The patient is trying to extend the wrist joint to the ulnar side against resistance



Fig 1347 Abductor pollicis longus The patient attempts to maintain his thumb in abduction against the examiner's resistance



Fig 1348 Extensor pollicis brevis The patient is resisting an attempt to flex the thumb at the metacarpophalangeal joint.

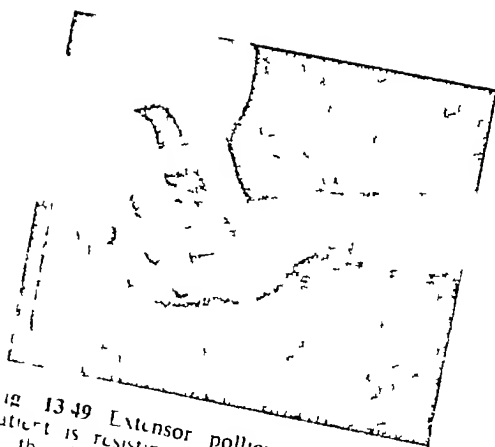


Fig 13.49 Extensor pollicis longus. The patient is resisting an attempt to flex the thumb at the interphalangeal joint



Fig 13.50 Pronator teres. With the arm extended by the side the patient resists an attempt to supinate the hand

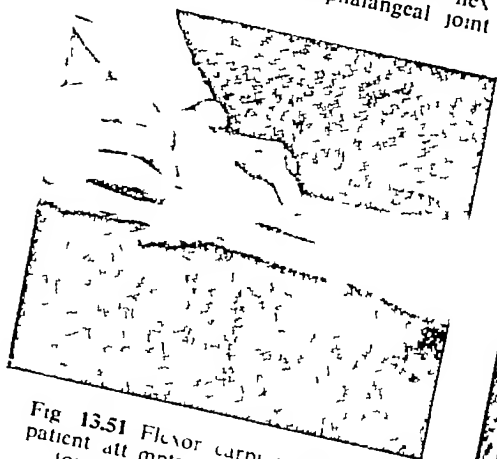


Fig 13.51 Flexor carpi radialis. The patient attempts to flex the wrist towards the radial side against resistance



Fig 13.52 Flexor digitorum sublimis. The patient is resisting an attempt to straighten the finger at its first interphalangeal joint while the proximal phalanx is fixed

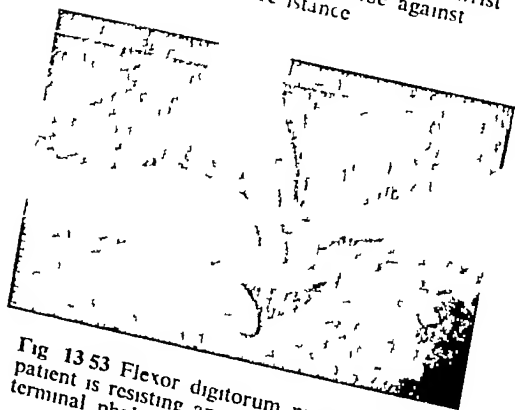


Fig 13.53 Flexor digitorum profundus. The patient is resisting an attempt to extend the terminal phalanx of the index finger while the second phalanx is fixed



Fig. 13.54 Flexor pollicis longus. The patient is resisting an attempt to extend the thumb while the proximal phalanx is fixed

is asked to abduct the thumb against resistance, in a plane at right angles to the palm (Fig 13 47)

**EXTENSOR POLLICIS BREVIS** (Radial nerve, C 7, C 8)

*Action* Extension of proximal phalanx of the thumb

*Test* The metacarpal of the thumb being stabilized, the patient tries to extend the proximal phalanx against resistance (Fig 13 48) The distal phalanx is kept in flexion, to minimize the action of extensor pollicis longus

**EXTENSOR POLLICIS LONGUS** (Radial nerve, C 7, C 8)

*Action* Adduction of the metacarpal of thumb and extension of distal phalanx of the thumb

*Test* Extension of distal phalanx against resistance (Fig 13 49) A classical feature of *radial nerve paralysis* is *wrist drop* Extension of the fingers at the interphalangeal joints can still be carried out, but extension of thumb is lost

**PRONATOR TERES** (Median nerve, C 6, C 7)

*Action* Pronation of forearm

*Test* The elbow is held by the side of the trunk and the forearm flexed at right angles Starting from a position of slight supination, patient either resists attempt to supinate the forearm, or tries to pronate the forearm against resistance (Fig 13 50)

**FLEXOR CARPI RADIALIS** (Median nerve, C 6, C 7, C 8)

*Action* Palmar flexion of hand at wrist, with radial deviation

*Test* Flexion of wrist radially against resistance, with the fingers kept relaxed (Fig 13 51)

**FLEXOR DIGITORUM SUBLIMIS** (Median nerve, C 7, C 8, T 1)

*Action* Flexion of middle phalanges of the fingers

*Test* With the proximal phalanx fixed, the patient resists attempt to straighten the finger at its proximal interphalangeal joint (Fig 13 52)

**FLEXOR DIGITORUM PROFUNDUS** (Radial portion median nerve, C 8, T 1, Ulnar portion ulnar nerve C 7, C 8, T 1)

*Action* Flexion of distal phalanges of fingers

*Test* With the proximal and middle phalanges fixed in extension, the patient tries to flex the distal phalanx or resists attempt to extend the distal phalanx (Fig 13 53) -

**FLEXOR POLLICIS LONGUS** (Median nerve, C 8, T 1)

*Action* Flexion of the distal phalanx of the thumb

*Test* The patient attempts to flex the terminal phalanx of the thumb against resistance, whilst the proximal phalanx is fixed (Fig 13 54)



**ABDUCTOR POLLICIS BREVIS** (Median nerve, C 8, T 1)

*Action* Palmar abduction of thumb

*Test* Abduction of the thumb against resistance perpendicular to the plane of the palm. The patient's thumb is so placed that the nail is in a plane at right angles to the palm, with a fountain pen between thumb and palm (Fig 13 55A). The patient tries against resistance to bring the edge of the thumb to a point vertically above its original position (Fig 13 55B).

**OPPONENS POLLICIS** (Median nerve, C 8, T 1)

*Action* Movement of the metacarpal of the thumb across the palm

*Test* Attempt to touch, against resistance, the tip of the little finger to the thumb, the thumb nail remaining in a plane parallel to the palm (Fig 13 56).

A complete median nerve palsy produces the ape (simian) hand with the thumb tending to lie in the same plane as the palm and atrophy of the thenar muscles. Inability to flex the distal phalanx of the thumb and index finger is tested by the patient trying to make a fist. The index finger sticks out as though in the act of pointing (See Fig 13 78).

**LUMBRICAL-INTEROSSEOUS MUSCLES** (Median and Ulnar nerves, C 8, T 1)

*Action* Extension of the interphalangeal joints and flexion of the metacarpophalangeal joints

*Test* With the metacarpophalangeal joint hyper-extended and fixed, the patient tries to extend the interphalangeal joint against resistance (Fig 13 57). Each finger should be tested separately.

**FLEXOR CARPI ULNARIS** (Ulnar nerve, C 7, C 8)

*Action* Flexion and ulnar deviation of hand

*Test* With the hand flat on the table, palm upwards (Fig 13 58A), the patient tries to abduct the little finger strongly (Fig 13 58B). The tendon of the flexor carpi ulnaris can be seen as the muscle comes into action (to fix the point of origin of abductor digiti minimi).

**FLEXOR DIGITORUM PROFUNDUS** (Ulnar portion, ulnar nerve, C 8, T 1)

*Action* Flexion of distal phalanges of fingers

*Test* The patient resists an attempt to extend the little finger at the distal interphalangeal joint, while the middle phalanx is fixed (Fig 13 59).

**ABDUCTOR DIGITI MINIMI** (Ulnar nerve, C 8, T 1)

*Action* Abduction of the little finger

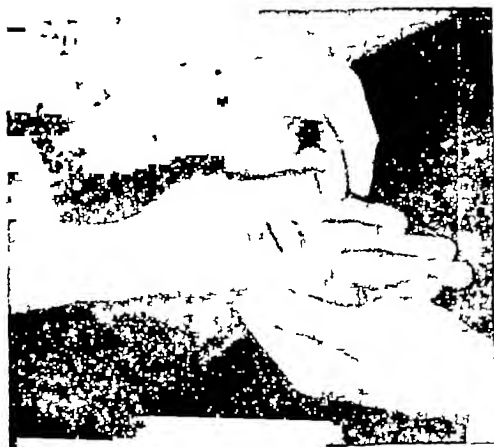
*Test* With the hand flat on the table, abduction of the little finger is carried out against resistance (Fig 13 60).



**Fig 13.55A Abductor pollicis brevis**  
An object such as a fountain pen is placed between the thumb and the base of the forefinger of the patient to prevent full adduction



**Fig 13.55B Abductor pollicis brevis**  
The patient attempts to raise against resistance the edge of the thumb vertically above the starting point



**Fig 13.56 Opponens pollicis** The patient attempts to touch the little finger with the thumb against resistance



**Fig 13.57 Lumbrical interosseous muscles** With the metacarpophalangeal joint hyperextended and fixed, the patient is trying to extend the distal interphalangeal joint against resistance



**Fig 13.58A Flexor carpi ulnaris** The patient's hand is kept flat on the table with palm upwards



**Fig 13.58B** The patient attempts to abduct the little finger

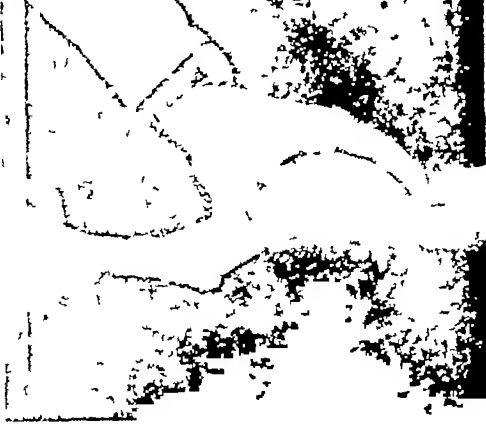
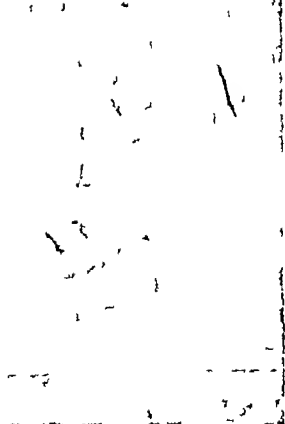


Fig. 13 60 Abductor digiti minimi With the back of the hand placed flat on the table the patient tries to abduct the little finger against resistance

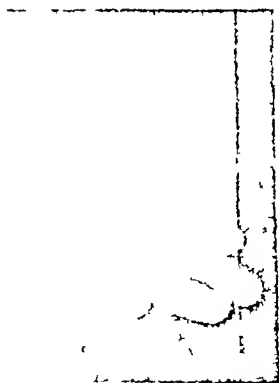


Fig. 13 62 First dorsal interosseous muscle With the hand placed flat on the table the patient attempts to abduct the index finger against resistance

Fig. 13 63 First palmar interosseous muscle With the palm placed flat on the table the patient is trying against resistance to bring the abducted index finger towards the median line.



Fig. 13 64 Adductor pollicis The patient holds against resistance a piece of wood between the thumb and the palmar aspects of the forefinger

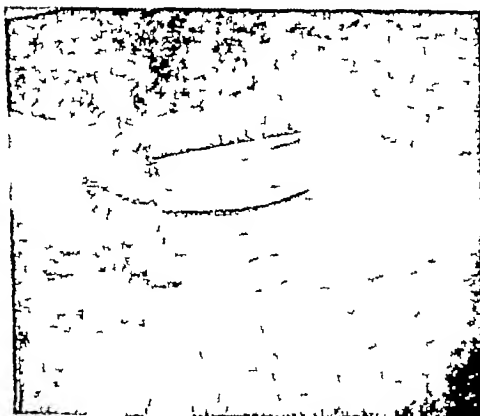


Fig. 13 65 Adductor pollicis The patient holds against resistance a piece of wood between the thumb and the palmar aspects of the forefinger

**OPPONENS DIGITI MINIMI (Ulnar nerve, C 8, T 1)**

*Action* Opposition of little finger towards thumb

*Test* Attempt to cup the palm and carry the fifth finger in front of the others (Fig 13 61)

**INTEROSSEI MUSCLES (Ulnar nerve, C 8, T 1)**

(1) *Dorsal interossei* *Action* Abduction of the index, middle and ring fingers away from the midline of the middle finger Double action on the middle finger, both radial and ulnar abduction, radial abduction of the index finger, ulnar abduction of the ring finger

*Test* To test the first dorsal interosseous muscle, with the fingers and palm flat on the table, the patient tries against resistance to abduct the index finger (Fig 13 62)

(2) *Palmar interossei* *Action* Adduction of the index, ring and little fingers towards the middle finger

*Test* Adduction of individual fingers against resistance with the fingers extended and kept flat on the table To test the first palmar interosseous muscle, the patient tries against resistance to bring the abducted index finger towards the median line (Fig 13 63) Adduction can be tested by retaining a strip of stiff paper between the fingers, as attempt is made to withdraw it

**ADDUCTOR POLLICIS (Ulnar nerve, C 8, T 1)**

*Action* Adduction of metacarpal of thumb

*Test* Retention of strip of paper between the thumb and palm against resistance, the thumb lying along the palmar aspect of the index finger and its nail in a plane at right angles to the palm (Fig 13 64)

*Forment's sign* In case of suspected ulnar palsy, the patient is asked to hold a piece of cardboard firmly with the thumb and index finger of each hand and then pull vigorously If flexion of the terminal phalanx of the thumb occurs on one side, the test is positive and indicates paralysis of ulnar nerve on that side

**ABDOMINAL MUSCLES (Upper T 6 to T 9, Lower T 10 to L 1)**

*Action* Abdominal compression

*Test* The patient, lying down, tries to flex the neck against resistance applied to the forehead Contraction of the abdominal muscles can be seen and felt If there is weakness of the lower abdominal muscles, the umbilicus will be seen to move upwards

**EXTENSORS OF BACK**

*Test* The patient lies prone, with hands clasped over buttocks, and attempts to elevate head and shoulders above the bed whilst the examiner holds the legs down

**ILIOPSOAS** (Femoral nerve, and L 1, L 2, L 3)

*Action* Flexion of the thigh at hip-joint

*Test* With the knee flexed and leg supported by the examiner, the thigh is sufficiently flexed as to make the angle between it and the trunk a right angle. The patient now tries to flex the hip-joint against resistance (Fig 13 65)

**SARTORIUS** (Femoral nerve, L 2, L 3)

*Action* Flexion of the knee joint

*Test* With the thigh laterally rotated, the patient tries to flex the knee against resistance. The muscle belly can be felt and sometimes seen (Fig 15 66)

**QUADRICEPS FEMORIS** (Femoral nerve, L 2, L 3, L 4)

*Action* Extension of leg at knee

*Test* The patient tries to extend the lower leg against resistance (Fig 13 67)

**GLUTEUS MAXIMUS** (Inferior gluteal nerve, L 5, S 1, S 2)

*Action* Extension of thigh at hip and lateral rotation of thigh.

*Test* The patient lies on his face and attempts to lift the knee off the bed against resistance. The muscle can be palpated and seen (Fig. 13 68)

**ADDUCTORS OF THIGH** (Obturator nerve, L 2, L 3 (Part of adductor magnus is supplied by the sciatic nerve))

*Action* Adduction of thigh (principally)

*Test* The patient holds the knees together while the examiner tries to separate them, or the patient lies down with the leg extended and tries to adduct it against resistance (Fig. 13 69)

**ABDUCTORS OF THIGH** (Gluteus medius, gluteus minimus and tensor fasciae lata) (Superior gluteal nerve, L 4, L 5, S 1)

*Action* Abduction of the thigh (with internal rotation)

*Test* The patient lies on his back with the knee extended and tries to abduct the limb against resistance (Fig 13 70). The muscle bellies can be felt and sometimes seen.

**MEDIAL ROTATORS OF THIGH** (Same as abductors)

*Test* The patient lies on his face with the knee flexed at right angles and tries to carry the foot laterally against resistance (Fig 13 71)

**LATERAL ROTATORS OF THIGH** (Gluteus maximus, obturator internus, quadratus femoris and gemelli) (L 4, L 5, S 1, S 2)

*Test* With the knee flexed to a right angle, the patient tries to rotate the thigh laterally against an attempt by the examiner to rotate it medially.



Fig 13.65 Iliopsoas The patient lying on his back attempts to flex his thigh against resistance



Fig 13.66 Sartorius The patient lies on his back with the hip laterally rotated and is trying to flex the knee against resistance



Fig 13.67 Quadriceps femoris The patient lying on his back attempts to extend the knee against resistance

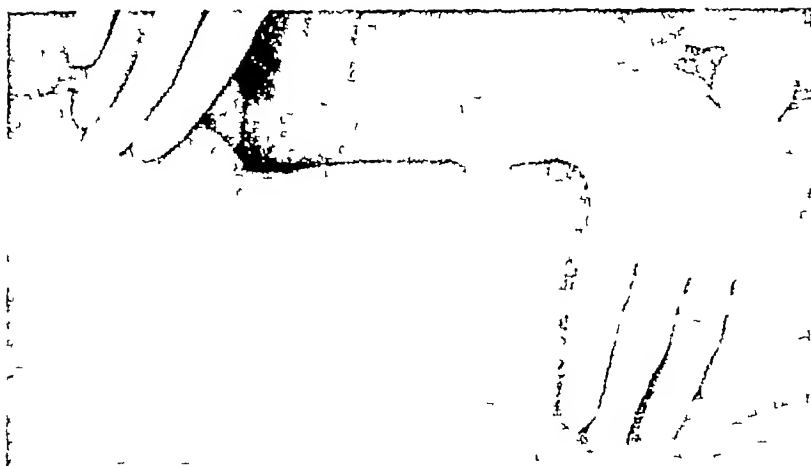


Fig 13.68 Gluteus maximus The patient lies on his face and attempts to lift the knee off the bed against resistance



Fig 13 69 Adductors of thigh  
The patient attempts to adduct  
the extended leg against  
resistance



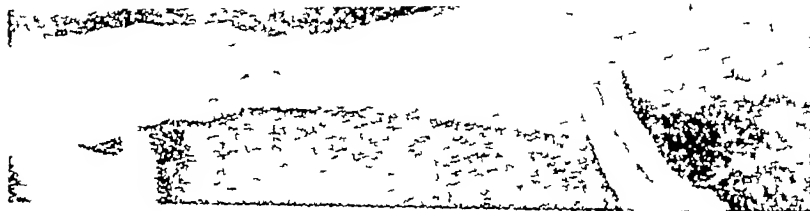
Fig 13 71 Medial rotators of  
thigh The patient lies on his  
face with the knee flexed to a  
right angle and attempts to  
force the foot outwards against  
resistance



Hamstring muscles  
The patient lies on his face and  
attempts to flex the knee  
The tendon  
of the biceps can be felt and  
seen



Fig 13 72B Ha Hamstring muscles  
The patient lies on his back and  
is trying to flex the knee  
The tendon  
of the biceps can be  
seen



mus The patient lies on the face and is attempting to plantar-flex the ankle joint against resistance

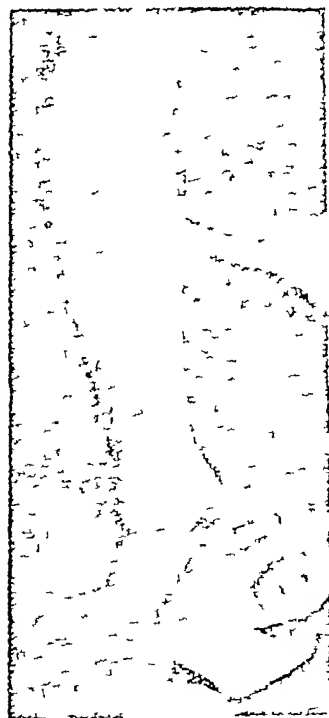


Fig 13 74 Tibialis posterior The patient plantar-flexes the foot slightly and then tries to invert it against resistance

Fig 13 75 Tibialis anterior The patient is trying to dorsiflex the ankle against resistance

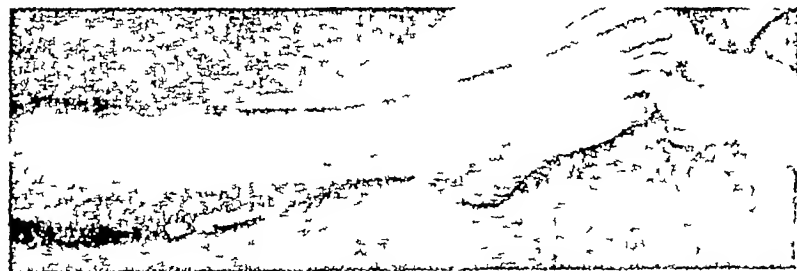


Fig 13 76 Long-flexors of foot. The patient attempts to flex the terminal phalanges of the toes against resistance



Fig 13 77 Extensor digitorum longus The patient flexes the terminal phalanges of the toes against resistance

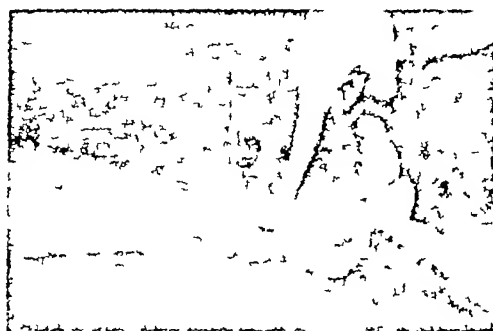


Fig 13 78 Extensor hallucis longus The patient attempts to dorsiflex the great toe against resistance



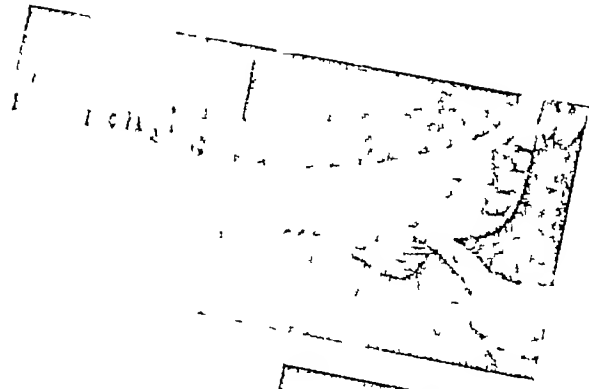


Fig. 13.79 Extensor digitorum brevis The patient is trying to dorsiflex the great toe against resistance

Fig. 13.80 Peroneus longus and patient trying to resist resistance

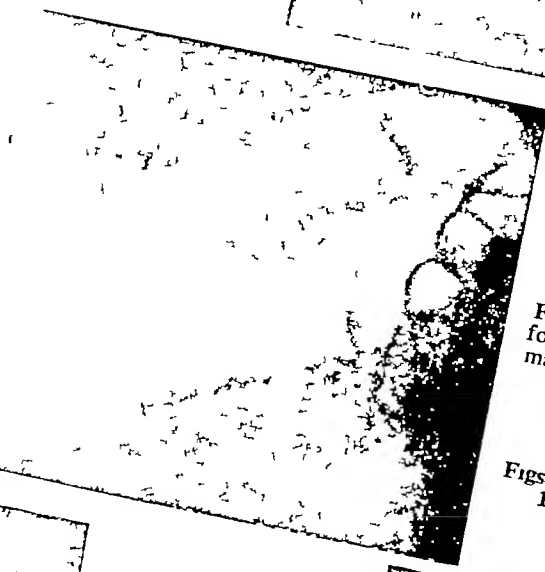


Fig. 13.81 Small muscles of foot The patient is trying to make a cup of the sole of the foot.

Figs. 13.83, 13.84, 13.85 and 13.86, see pp 496-503



Fig. 13.82 Athetoid movement

Fig. 13.87 Enlargement of great auricular nerve in leprosy



**HAMSTRINGS** (Sciatic nerve, L 4, L 5, S 1, S 2)

*Action* Flexion of leg at knee

*Test* The patient lies on his face and tries to flex the knee against resistance. The tendons of the biceps laterally (Fig 13 72A) and the semitendinosus medially (Fig 13 72B) can be felt and seen. Or the patient may lie prone with the knee partly flexed and try to flex it further against resistance.

**GASTROCNEMIUS** (Medial popliteal nerve, S 1, S 2)

*Action* Plantar flexion of foot

*Test* Plantar flexion of foot against resistance (Fig 13 73)

**TIBIALIS POSTERIOR** (Sciatic nerve, L 4, L 5)

*Action* Inversion of foot

*Test* The patient lies on his back and tries to invert the plantar-flexed foot against resistance (Fig 13 74)

**TIBIALIS ANTERIOR** (Lateral popliteal nerve, L 4, L 5)

*Action* Dorsiflexion and inversion of foot

*Test* Dorsiflexion of foot against resistance applied to the dorsum of foot (Fig 13 75)

**LONG FLEXORS OF FOOT** (Flexor digitorum longus, flexor hallucis longus) (Medial popliteal nerve, S 1, S 2)

*Action* Plantar flexion of the toes, especially at terminal interphalangeal joints

*Test* Plantar flexion of distal joints of the toes against slight resistance (Fig 13 76)

**EXTENSOR DIGITORUM LONGUS** (Lat popliteal nerve, L 5, S 1)

*Action* Dorsiflexion of toes

*Test* Dorsiflexion of toes against resistance (Fig 13 77)

**EXTENSOR HALLUCIS LONGUS** (Lat popliteal nerve, L 5, S 1)

*Action* Extension of great toe

*Test* Dorsiflexion of great toe against resistance, the foot being kept in a neutral position (Fig 13 78)

**EXTENSOR DIGITORUM BREVIS** (Lat popliteal nerve, S 1)

*Action* Dorsiflexion of the toes

*Test* Dorsiflexion of great toe against resistance (Fig 13 79)

**PERONEUS LONGUS AND BREVIS** (Lateral popliteal nerve, L 5, S 1)

*Action* Eversion of foot, kept in plantar-flexed position, against resistance applied to lateral border of foot (Fig 13 80)

**SMALL (INTRINSIC) MUSCLES OF FOOT** (Medial popliteal nerve, S 1, S 2)

*Action* Fanning out and bringing together of the toes and flexion of proximal phalanges of the toes

*Test* Attempt at "cupping" of the sole of the foot (Fig 13 81)

Since the testing of individual muscles is time consuming and unnecessary in every neurological patient, it is easier to think in terms of movements and not individual muscles and a selected group of muscles may be tested the rhomboids, deltoids, flexors and extensors of the elbow, wrist and fingers, the interossei, and then the abdominal muscles. This is followed by examination of flexors of the hip, quadriceps, dorsiflexors and plantarflexors of the foot, and flexors and extensors of the toes. The extensors of the hip and flexors of the knee are then tested by making the patient lie in a prone position.

**Motor weakness or paralysis** Investigation of various muscles and muscle groups for motor power may bring to light complete or partial loss of power in some part or other of the body. Partial loss of muscle power or muscle weakness is described as paresis and complete or total loss of power is labelled paralysis. However, the term paralysis is often used to signify partial as well as complete palsy. Paralysis should be considered apart from the state of general weakness or debility.

Paralysis can result from a lesion in any part of the motor system from the precentral convolution in the cerebral cortex to the muscle. Loss of muscle power need not be in terms of an entire limb or side of body, but may affect single muscle groups or individual muscles, depending on the site and size of the causative lesion. The diagnostic consideration of paralysis can be simplified by the following subdivisions which relate to the location and distribution of weakness.

**MONOPLÉGIA** One limb, either lower (crural monoplegia) or upper (brachial monoplegia), weak or paralyzed.

**HEMIPLEGIA** This is the most commonly occurring paralysis and refers to loss of strength on one side of the body, including often the face.

**PARAPLEGIA** Symmetrical paralysis of both lower extremities mostly due to disease of the spinal cord. A paraplegia may be paraplegia in extension if the transverse lesion involves the pyramidal tract, paraplegia in flexion is indicative of a more severe lesion due to both pyramidal and extrapyramidal involvement.

**QUADRIPLEGIA OR TETRAPLEGIA** This term signifies weakness of all four extremities due to a severe or long standing affection.

**DIPLEGIA** A paralysis of any two extremities, usually both legs, is referred to as diplegia. By convention diplegia is cerebral in origin.

It should be remembered that the terms diplegia, hemiplegia, paraplegia, etc are all used in relation to pyramidal damage and, strictly speaking, not for lower motor neurone damage

**ISOLATED PARALYSIS** This indicates weakness localized to one or more muscle groups

## COORDINATION /

The harmonious activity and correct sequence of action of appropriate muscles, inherent in a normal movement or reaction, are collectively referred to as coordination. Lack of coordination of muscles is referred to as incoordination, ataxia or asynergia ✓

The coordination of bodily musculature depends on three principal systems, viz, (1) the afferent system, subserving postural sense, (2) the vestibular system, orientating the position of body in space, and (3) the cerebellar system, which maintains a smooth and balanced flow of impulses to the muscles. A lesion in any of these systems will give rise to imperfect coordination or incoordination. It should be remembered, however, that a retardation or weakness of movement from pyramidal or extrapyramidal disturbance may render coordination difficult

**Tests of function** The following tests are recommended for the investigation of muscle coordination in different parts of the body

**UPPER EXTREMITY** (a) Outstretched arms test As a preliminary observation or screening test, ask the patient to hold the outstretched arms, with wrists slightly dorsiflexed, in front of him. Observe (i) if patient has any difficulty in maintaining the position. A drift of the affected arm (with at times a mild drifting apart of the affected fingers) suggests an early pyramidal tract motor defect. (ii) Ask the patient to close his eyes and observe if the hands remain level. In cerebellar disease one hand may rise up and oscillate. (iii) Tap each arm sharply. If it tends to fall it suggests hypotonia of cerebellar disease, defective sensation or weakness. In cerebellar disease the arm on the affected side tends to oscillate and returns to its original position after several bounces

(b) Finger-nose test The patient is asked to fully extend and abduct the arm and then touch the tip of his nose with the fore-finger, the hand being in the mid-prone position as the index finger approaches the nose. The test is repeated with the eyes closed. Observe if the finger accurately touches the tip of the nose and if there is any tremor. Abnormalities (i) In cerebellar disease, the finger moves to the nose in a wavering but not jerky fashion and is brought to touch the nose fairly accurately at the end. With intention tremor with which it may be confused, on the other hand, a side-to-side oscillation develops as the finger approaches the nose. (ii) In postural ataxia, ✓

with eyes open, the movement tends to be smooth but the finger hesitates before finally touching the nose. With eyes closed, the finger cannot find the nose and touches some part of the face first and the finger is then dragged along the face to the nose.

(c) *Nose-finger-nose test* The patient places the tip of the index finger on his own nose, then reaches to touch the tip of the examiner's finger, and again touches the tip of his own nose. The test should be repeated at different speeds, the examiner holding his finger in a different position for each movement.

(d) *Finger to finger test* The patient is asked to bring his arms to the sides and then bring the tips of the index fingers together through a forward arc.

(e) *Finger-thumb test* (Thumb tap test) The patient is asked to approximate the tip of the index finger to the top of the thumb of the same hand in rapid succession. The normal patient will move the other digits as the finger taps, but there will be no movement at the wrist, elbow, or shoulder.

(f) *Pronation-supination test* The patient is asked to sit up and pat his knee alternately with the palm and dorsum of one hand. The test is then repeated on the other side and finally on both sides simultaneously.

(g) *Pointing and past pointing test* It is useful especially for testing disturbance of vestibular mechanism. The patient is asked to move the forward-extended arm with the index finger pointing upward, to the vertical position and bring it directly down and touch the examiner's index finger which is held at the point where the patient began the movement. After this is done a few times with the eyes open, the patient is asked to close his eyes and repeat the movement. In vestibular disease, the patient will past-point with both hands in the direction of the involved side. In unilateral cerebellar disease, the patient past-points toward the side of the lesion, but only with the ipsilateral arm.

(h) *Threading a needle* In the case of a female patient, it is worthwhile asking her to thread a needle.

(i) *Buttoning and unbuttoning test* The patient is asked to button his coat or shirt and then unbutton it. The test may be repeated once or twice, if required.

(j) *Writing test* The patient is asked to write his name and address on a piece of paper with a pencil.

(k) *Drinking test* The patient is asked to lift a glass, filled to the brim with water, from the table to his lips for drinking.

(l) *Stopping at fixed points on a straight line* Another test for dysmetria consists in having the patient stopping at fixed points on a drawn straight line. He may have difficulty in starting at the correct point and may either stop before he reaches the second point or overshoot the mark.

(m) *Rapid hand tapping* The back of one hand is tapped rapidly with the fingers of the other. In cerebellar disease the tap becomes a rotatory stroking movement.

LOWER EXTREMITY (a) Screening test Wriggling of the toes corresponds to the outstretched arms test, and inability to wriggle the toes is likewise a sign of cerebrospinal dysfunction. For examining the lower extremities for drift, the patient is made to lie face down and the legs flexed at the knee at about  $45^\circ$

(b) Finger-toe test The recumbent patient is asked to touch the examiner's finger, held about 2 feet away, with the big toe. After the finger has been touched, it is moved to another position and the patient asked to once again push the examiner's finger with his toe. Normally the patient will be able to do this with only a slight bounce at the end of the movement.

(c) Heel-knee test The patient is asked to touch his knee with the heel of the other leg, and then slide the heel down the shin and dorsum towards the big toe, without permitting the heel to slide off the leg. The foot should not be rubbed up and down the shin. The patient may then be asked to slide the heel back up the leg, if so desired. In cerebellar disease, the heel is carried up to overshoot the knee. As the heel is carried downward, it begins to execute an action tremor. The patient with posterior column disease may likewise miss the knee because of the inability to recognize position in space, when he moves the heel down the shin, the heel tends to fall to one or other side or may slide off the leg entirely.

(d) Writing in the air The patient may be asked to write out simple figures, such as the figure 8 in the air with the foot, the eyes being kept shut.

(e) Romberg's test The patient stands with his feet close together and eyes open. After he has assumed a stable position he is asked to close his eyes. When the sign is positive, the patient begins to sway and may even fall over if not supported. In early cases, the patient may be asked to stand on the tips of his toes with knees semiflexed, and then ordered to shut the eyes. Rombergism is characteristic of proprioceptive deficiency and occurs in tabes dorsalis, subacute combined degeneration, sensory polyneuropathies and posterior cord compression at a high level. It is also present in labyrinthine disorders. It should be remembered that slight swaying with the eyes closed is normal.

CT

#### Special tests for cerebellar incoordination

(1) Dysmetria Inability to stop an intended movement at the correct time and place with resultant overshooting is referred to as dysmetria. It is best revealed by the finger-nose test. The finger is pushed beyond the nose to the cheek, or strikes the nose with force. At times, the patient may attempt to correct his defect by arresting suddenly the movement of his finger before it reaches the nose, and then complete the movement by slowly bringing the finger to the tip of the nose.

(2) Dysdiadokokinesia The ability to carry out rapid pronation and supination of the forearm (adiadokokinesia) on each side is tested in turn. In

cerebellar disease, this movement is carried out slowly, and somewhat irregularly or clumsily (dysdiadokokinesia). Other tests for the same phenomenon include a rapid opening and closing of the fist, repeated tapping of the foot on the floor, and touching the tip of each finger to the thumb.

(3) *Rebound phenomenon* The patient flexes his arm at the elbow and is asked to pull the forearm towards the shoulder against resistance. The examiner then suddenly releases the resistance. In cerebellar disease, the patient's hand flies up with considerable force and may strike the face. Normally, such a movement is arrested almost immediately with or without a recoil.

(4) *Pointing test* The patient is asked to touch with his index finger the examiner's finger held in front of him. He then closes his eyes, drops his hand and arm, and again tries to bring back his index finger to the examiner's finger which is held in position. In vestibular involvement, the patient will constantly deviate (past-point), with both hands, in the direction of the side of involvement. In unilateral cerebellar disease, the patient past-points towards the side of the lesion, with the arm on the side of affection but not with the contra-lateral arm. This is a test of vestibular function rather than of pure cerebellar tract disease.

(5) *Walking along a straight line* In a one-sided cerebellar lesion, the patient, during this test, tends to deviate towards the affected side.

(6) *Decomposition of movement* Instead of carrying out a single well-defined movement with a limb, the movement is irregularly split-up or disorganized into a succession of movements, such as flexion and extension at the various joints.

## INVOLUNTARY MOVEMENTS

In health, the body and limbs are at rest, there being no movements except when voluntarily induced. Involuntary movements, which may occur during consciousness or unconsciousness, are abnormal and indicative of disease.

Abnormal involuntary movements are a conspicuous feature of certain neurological diseases, and a correct clinical diagnosis may depend on a careful study of such movements. Lesions affecting peripheral nerves, spinal cord and various centres of the brain-stem, all have their own characteristic forms of involuntary motor activity. It should be remembered, however, that involuntary movements are merely symptoms, and as such may occur in a variety of different diseases. Hence, in addition to determining the exact nature of the involuntary movement, all clinical data relevant to the case, such as the onset, course, and existence of associated features, should be carefully considered.

Since normal control of muscle activity implies both, the power of muscle relaxation as well as that of contraction, in most cases of involuntary move-

ment the abnormal involuntary motor activity has to be regarded as a "release" phenomenon resulting from loss of inhibitory control of a "higher" controlling centre. All involuntary movements with the exception of tics and habit spasms are indicative of disease. The features to be observed in connection with the involuntary movement are situation, muscle groups involved, character, whether at rest or on movement or on assuming a particular posture, rhythmic or arrhythmic, and the effects of emotion, sleep, and of voluntary movements on these involuntary movements.

Certain varieties of involuntary movements are of particular clinical importance. *True* involuntary movements (in order of extent, i.e., from small to large movements) are muscle fasciculations, tremors (fine, medium or coarse), choreiform movements, athetotic movements, myoclonus, torsion spasm, and hemiballismus. Other varieties of ill-defined involuntary movements are habit spasms (tics), cramps and spasms (several varieties).

(1) **Muscle fasciculation** Irregular, intermittent, flickering involuntary contractions of muscle fibres, visible through the skin, and initiated or increased by mechanical stimulation, percussion of muscle, voluntary over-breathing, fatigue or cold, are referred to as muscle fasciculation. Muscle fasciculation, although observed rarely in normal healthy subjects (often following exertion), is usually indicative of active degeneration of anterior horn cell, as in progressive muscular atrophy, amyotrophic lateral sclerosis. It may also occur in lesions of anterior roots (cervical spondylosis) and peripheral neuropathies (usually chronic). It is well to search for fasciculations in a well-lighted room. It may prove necessary at times to percuss or flick a muscle lightly in order to activate these muscle twitchings. Widespread fasciculations can be benign but usually indicate active anterior horn cell disease.

*Muscle fibrillations* are really never seen with the naked eye and are only recordable on electromyography. The word fibrillation is frequently used wrongly and synonymously with fasciculation. Although both phenomena arise as the result of muscle degeneration, fasciculations are visible to the naked eye, whilst fibrillations are not, except in the tongue.

(2) **Tremor.** One of the commonest varieties of involuntary movements, a tremor is a to-and-fro, rhythmical oscillation of the fingers of the outstretched hand, or of the tongue, head, lips or eyelids. Due to alternating contractions of opposing groups of muscles, tremors differ widely as to rate (rapid or slow), rhythm, amplitude (low in fine tremors and high in coarse), character (to-and-fro, rotatory, pill-rolling or trombone-type), duration (transitory, temporary or persistent), extent (localized or widespread), relation to activity and rest ("intention" tremor, action tremor or tremor of rest), relation to sleep (continuing or ceasing during sleep) and relation to emotion or exercise.

*Fine tremor* (Toxic tremor) A fine but fast tremor is commonly associated with thyrotoxicosis, uraemia, anxiety or tension states, chronic alcoholism,



excessive smoking, amphetamine addiction, poisoning by mercury, lead, bismuth, arsenic, dilantin or barbiturates, frontal lobe tumour or hysteria, it may also occur as a familial characteristic

*Parkinsonian tremor* (Coarse tremor) This is a characteristic, to-and-fro, coarse, slow and rhythmic rotatory ("pill-rolling" or "cigarette-rolling") tremor, more marked at rest, frequently inhibited by voluntary movement or sleep. It may or may not be associated with rigidity. It is common in Parkinsonian syndrome, hepatolenticular degeneration (Wilson's disease), basal ganglia tumours, after head injury, cerebral arteriosclerosis, post-infective encephalomyelitis and manganese or carbon monoxide poisoning.

*Medium tremor* Intermediate in amplitude and rate between fine and coarse, a "medium" or moderately fine tremor is encountered in old age, general paralysis of the insane, uraemia, cholaemia, opium or mercury poisoning or delirium tremens.

*Intention tremor* (Action tremor) A tremor, absent at rest, brought out by voluntary movement and intensified towards the end of the movement is called an intention or action or cerebellar tremor. It is characteristic of diseases of cerebellum and spinocerebellar tracts, such as disseminated sclerosis, Friedreich's ataxia, tumour of the cerebellopontine angle and cerebellar atrophy.

*Hysterical tremor* Characterized by its variability, inconstancy, bizarre nature, lack of associated signs of organic involvement and aggravation in the presence of bystanders, a hysterical tremor may be either fine, medium or coarse.

*Physiological tremor* Transient tremor induced by extreme fatigue, emotional stress, such as fright, and shivering through cold.

*Wrist-flapping tremor* (Asterixis) In hepatocellular failure, a characteristic abnormality is the flapping tremor. It can be demonstrated by asking the patient to out-stretch his arms with the fingers extended. The rapid flexion-extension movements at the metacarpo-phalangeal and wrist joints are often accompanied by lateral movements of the digits.

*Batwing tremor* This is the most extreme degree of terminal intention tremor sometimes seen in Wilson's disease. The oscillations are violent and occur over a wide range.

*Perioral tremor* A coarse tremor confined to the orbicularis oris and the chin may be seen in general paralysis of the insane.

*Familial essential tremor* Familial tremors are absent at rest, appear when the arms are outstretched and may be intensified by movement e.g. writing. There is no associated rigidity or other evidence of organic disease. At autopsy, lesions have been found in the cerebellum.

*Postural tremor* Tremor of the outstretched hands often increased on volition is characteristic of lesions of the dentato-rubral-thalamic pathway.

(3) **Choreiform movements (Chorea)** Sudden, jerky, brief, unexpected, irregular, pleomorphic involuntary movements of muscles or muscle groups of the extremities, face or body are called choreiform movements. They are encountered most often in rheumatic chorea (Sydenham's chorea). Whilst in a mild case the child may just appear fidgety, nervous or clumsy, in a severe case the involuntary jerks may be severe enough to throw the patient bodily out of bed, as in the chorea gravidarum of pregnancy. Other causes of choreiform movements are Huntington's chorea (a familial disease starting in middle age and associated with progressive dementia), senile chorea (of old age) and encephalitis lethargica. Chorea is usually accompanied by hypotonia, especially in Sydenham's chorea. The lesions are mainly in the caudate nucleus and putamen.

(4) **Athetotic movements (Mobile spasm)** (Fig 13.82) Athetoid movements are slow, smooth, contortive, sinuous or writhing involuntary movements of the extremities, particularly of distal parts, and often aggravated by emotion and voluntary movements. More common in children than adults, athetosis may occur in congenital athetosis, dyskinetic cerebral palsy, infantile hemiplegia, or degenerative disorders.

*Choreo-athetoid movements* are movements which have both slow and quick elements and either may predominate. Like athetosis they are seen with lesions of the corpus striatum.

(5) **Myoclonus.** Myoclonus is a single jerky movement of a muscle or limb, especially at a large joint. It may be repetitive, occurring several times a day for many years. A physiological form may occur in the light stages of sleep, the patient waking up suddenly because of violent myoclonic movement of a limb, mostly of the leg. According to some, nocturnal myoclonus is common in potential epileptics.

Myoclonus during waking is of uncertain etiology, and may be due to epilepsy (with the electro-encephalogram suggestive of epilepsy), inclusion body encephalitis of childhood or of familial origin. In some cases the aetiology is obscure.

*Palatal myoclonus* is a rapid and regular tremor of the uvula, with or without accompanying tremors of the pharynx, larynx and face on one side. Though it may occur rarely in elderly subjects free from disease, it is usually diagnostic of degeneration of the olive.

*Hiccough* is a myoclonic spasm of the diaphragm.

(6) **Torsion spasm (Dystonia)** Slow, rhythmical, regular and extensive involuntary movements of contortive type affecting the limbs and trunk and producing, when severe, grotesque postures. Occurring usually in cases of encephalitis lethargica, these are called torsion spasms. Spasmodic torticollis is the most commonly observed form of torsion spasm. Lesions are seen in the striation.

(7) **Hemiballismus** A violent flinging movement, confined to one side of the body, may be met with in lesions of the contralateral subthalamic region (vascular or encephalitic)

(8) **Other involuntary movements** (a) **Tics (*Habit spasms*)** A sudden rapid, coordinated and purposive, intermittent and repetitive involuntary movement, unchanging in character and location, increased by emotion and disappearing during sleep, is called a habit spasm or tic. Tics may take many forms, such as the frequent blinking of an eyelid, rolling of the eyeball, contortive twisting of the neck or a facial grimace. In severe cases, it may be widespread and associated with foul utterances (colorolalia) or complicated movements. Simple, psychical, coordinated and convulsive forms of tics have been described.

(b) **CRAMPS** Cramps are severe and painful involuntary contractions of large muscles, frequently of the calves. They may be caused by fatigue, over-use, exposure to cold, awkward posture, neuritis, deficiency of calcium or of Vitamin B<sub>1</sub>, dehydration, prolonged restriction of salt in the diet or hypokalaemia. They may be complained of by patients with amyotrophic lateral sclerosis.

(c) **SPASMS** The term spasm is used to describe a variety of muscular contractions, almost always involuntary, which frequently results in a movement. Sometimes a spasm is protective or reflex in nature and limits motion, as in the case of lumbar muscle spasm accompanying protrusion of a lumbar disc. Spasms may be of several types.

*Clonic spasms* are repetitive contractions rapid in onset, brief in duration and usually seen in epileptiform attacks.

*Tonic spasms* are more prolonged or continuous muscular contractions which may be brief in duration as in epilepsy or prolonged as in tetanus.

*Tetanospasm* The continuous spasms of tetanus cause trismus and retraction of the head. Superimposed on this, often as the result of external stimulation, are convulsive and tonic spasms resulting in flexion of upper extremities and extension of lower extremities and back, strychnine spasms are of somewhat similar nature.

*Spasm of tetany* Hypocalcemia and alkalosis may result in tetany or carpopedal spasm. The increased excitability of the peripheral nerves in this disease is demonstrable through certain clinical tests, useful for the detection of latent tetany.

Chovstek's sign consists of a brisk contraction of the facial muscles on tapping the facial nerve in front of the external auditory meatus.

Trousseau's sign is elicited by applying pressure, with the cuff of a sphygmomanometer, around the arm. An attack of carpal spasm indicates a positive result.

*Facial spasms* A variety of spasm affecting the facial musculature. Hemifacial spasm is characterized by brief tonic spasm of musculature of one half of the face. Blepharospasm is a reflex spasm of the eyelids associated with painful conditions of the eyes.

*Occupational spasm* A disorder of psychogenic origin, fairly common in those who have to use their hands persistently in certain stereotype types of movements. The spasm of the muscles may be severe and painful enough to prevent the individual from continuing the job. Many varieties of occupational spasm have been described, such as writer's, typist's, telegraphist's and piano-player's cramps.

*Oculogyric spasm* This is characterized by a tonic spasm of extra-ocular muscles, the eyes being usually turned upwards for seconds, minutes or hours. Such attacks were said to be characteristic of postencephalitic Parkinsonism, but can also be drug (phenothiazines) induced.

(d) DRUG INDUCED DYSKINESIAS. Drugs of the phenothiazine group and L-Dopa can produce dyskinesias which are mainly orofacial. Chorea has been reported with the contraceptive pill. Hence when confronted with involuntary movements or Parkinsonian syndrome, a history of drug administration must be ascertained.

## SENSORY SYSTEM

Under normal conditions, all sensations depend on impulses arising through adequate stimulation of end organs or receptors, and conveyed to the central nervous system through afferent or sensory fibres. A clue to the nature of sensory involvement can be frequently gained from the patient's description of his symptoms of pain, paraesthesia, numbness or tingling.

*Anatomy (Fig. 13.82)* *Tactile impulses* are conveyed to the brain by both short and long fibres of the posterior root system. Some fibres ascend through the posterior column of the same side, enter the nucleus gracilis and nucleus cuneatus, cross over to the opposite side and then ascend in the medial lemniscus to the ventrolateral thalamus. The great majority of fibres, however, after ascending or descending through several segments of the cord, cross over to enter and ascend along the anterior spinothalamic tract. In the posterior columns the most medially situated fibres convey impulses from the lower limbs and the most lateral from the upper. Touch, pressure, vibrations, joint sense and discrimination are mediated by the posterior columns.

*Temperature and pain* sensations are at first carried by the short posterior root fibres to terminate within the posterior horns at the levels of root-entry, two to four segments higher on the same side. After synapsing, the fibres cross over to the opposite side to join the lateral spinothalamic tract, ascend and terminate in the posterolateral ventral nucleus of the thalamus.

*Deep sensibility*, such as appreciation of position and movements, is carried by long posterior root fibres, which ascend along the ipsilateral posterior column to terminate round about the gracile and cuneate nuclei. From here, fibres cross over to the opposite side, and

ascend within the medial lemniscus to the postero-lateral ventral nucleus of the thalamus. A new relay of fibres then convey the impulses to the post-central gyrus of the parietal lobe of the cerebral cortex.

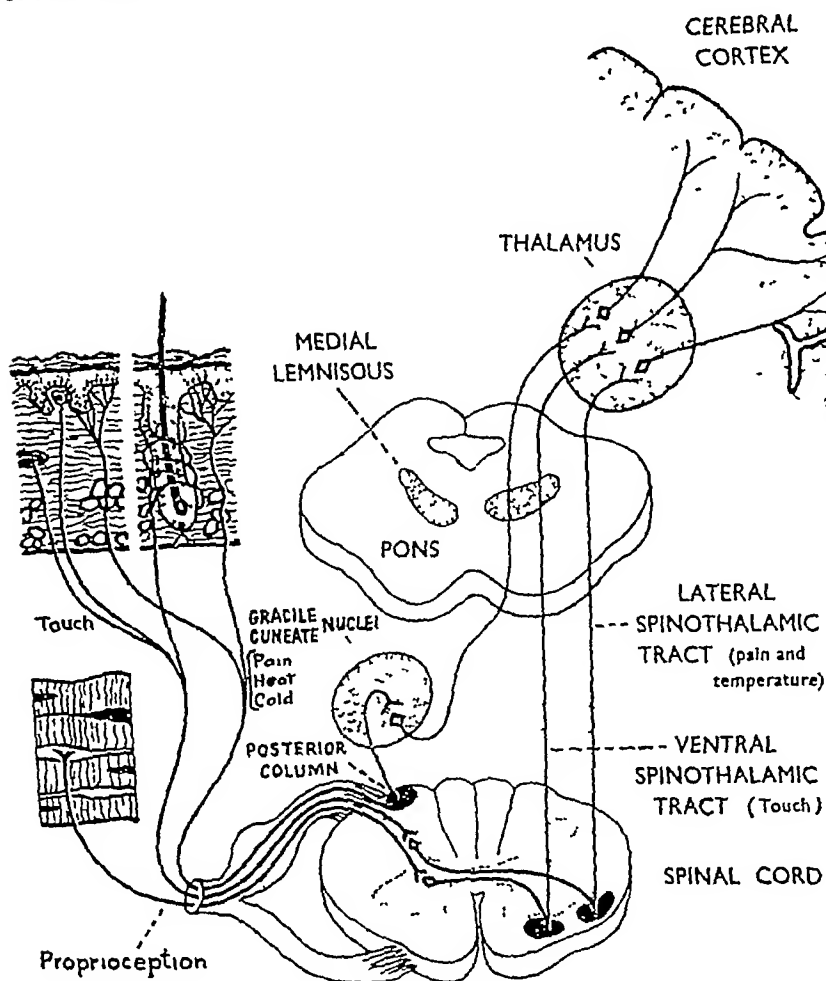


Fig 13 83 Diagram showing sensory pathways

The ventral posterior lateral nucleus of the thalamus is the final relay station in the sensory pathway and the ventral posterior medial nucleus for the trigeminal. Thalamocortical projections then travel to the somatosensory cortical area situated chiefly in the postcentral gyrus where there is somatopic representation of the various parts of the opposite side.

Many peripheral impulses, originating in deep structures, such as muscles, tendons and joints, and concerned with reflex activity, but not consciously perceived, are transmitted to the cerebellum, through the spinocerebellar tracts and posterior column.

**Sensory dermatomes** The cutaneous radicular and peripheral nerves are indicated in Figs 13 84, 13 85. It will be seen that there is significant

difference in the radicular and peripheral nerve distribution. As to the radicular fields, each spreads out on either side of the line of respective segment number for a sufficient distance to overlap with the adjoining two radicular fields. The overlap is such that anaesthesia can occur if two or more posterior roots are damaged, hyperaesthesia however does not occur. The rule that anaesthesia does not result from destruction of the posterior roots does not apply to irritative phenomena causing spontaneous pain which they can occur with only one root involvement.

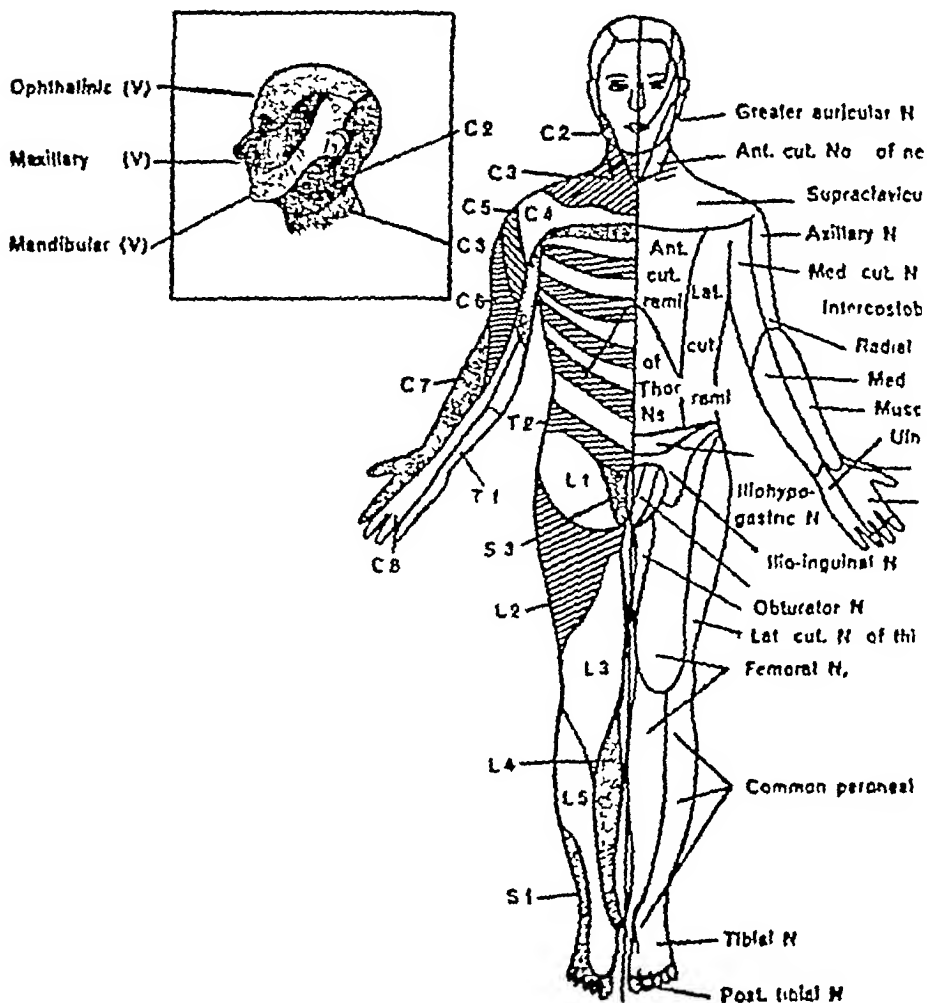


Fig 13.84 Cutaneous areas of distribution of spinal segments and peripheral nerves. Anterior aspect

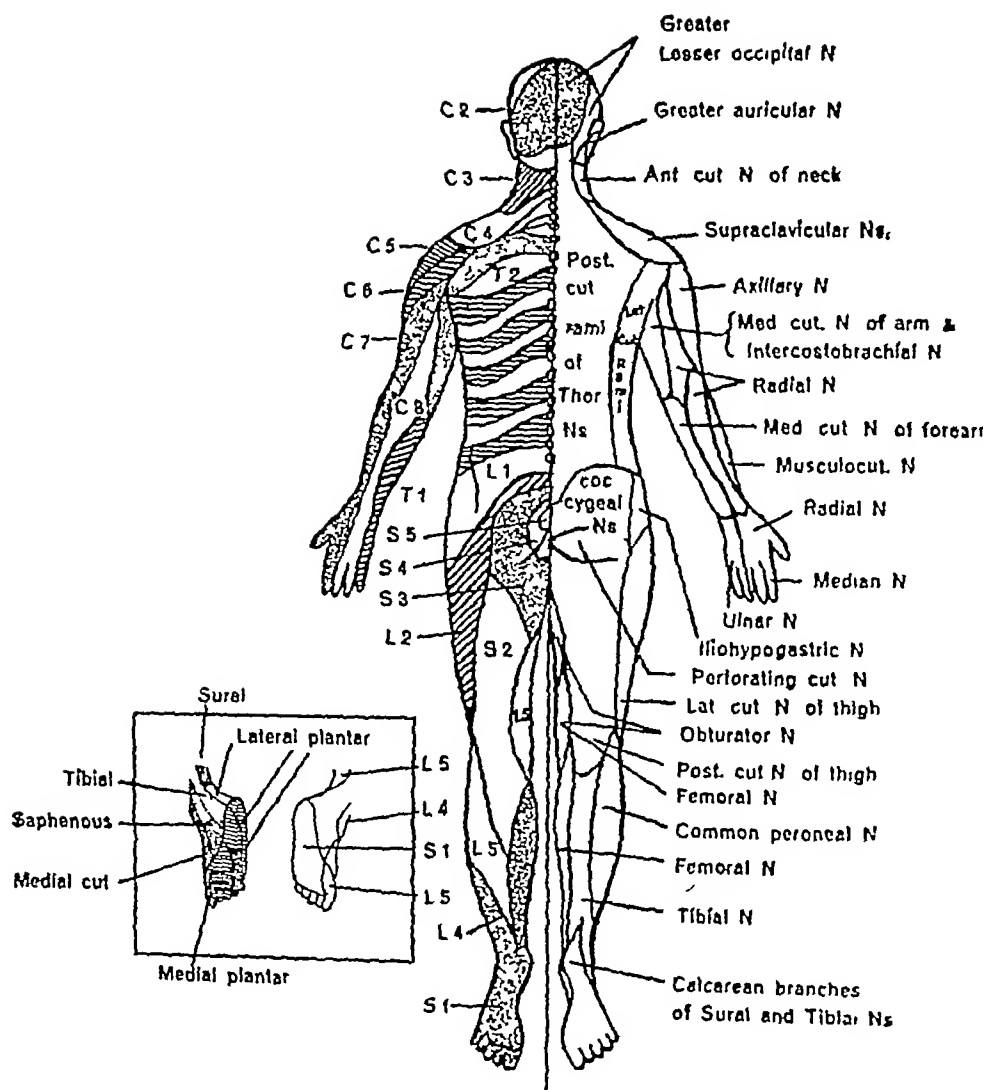


Fig. 13 85 Cutaneous areas of distribution of spinal segments and peripheral nerves Posterior aspect

For ready localization of the level of spinal cord (or the posterior roots) involved in case of disturbances of sensibility, the following points are worth remembering

The occiput is supplied by segment C3

The outer aspect of the shoulder is supplied by C5.

C7, the longest cervical spinal process supplies the middle finger (longest finger)

The scapulo digital line corresponds to upper limit of T1

T3 dermatome is situated in the axilla

T4 segment corresponds to the nipple (in the male)

T8 supplies the rib margin, T10 the umbilicus and T12 the pubis

Segment L1 corresponds to the ilio-inguinal line

L3 is situated at level of knee joint

S1 goes to the little toe

S3 dermatome corresponds to the perianal line

S3, S4, S5 are in concentric rings round the anus

**Sensory symptoms.** A sensory symptom described by the patient as an abnormal painful sensation induced by light touch is called *dysaesthesia*. *Paraesthesia* (perverted sensation) is an unusual sensation which may be described as pricking, tingling, numbness, burning, formication, which is spontaneous in origin

**Anaesthesia** By this is usually meant an impairment or loss of all forms of sensibility. It is customary to speak of light, moderate or total anaesthesia. When referring to special or selective forms of anaesthesia, viz., loss of tactile sense, pain sense and temperature sense, it is better to use the terms tactile anaesthesia, analgesia and thermoanaesthesia, respectively. Whilst the term *analgesia* refers to loss of pain sensibility, the terms *hypoalgesia* and *hyperalgesia* are used when the sensation of pain is diminished or exaggerated.

**Hyperaesthesia** This refers to an exaggerated perception of sensation such as touch, in hyperpathia a painful stimulus evokes intense pain or a reaction much greater than normal.

**Root pains** Disease processes, involving root fibres may induce spontaneous severe pains often of cutting or tearing or throbbing nature. Such pains are present in the early stages when damage is minimal and are projected to the periphery, to the area of skin in which the sensory end organs of the irritated fibres are situated. A patient having such pains is best able to indicate their situation when an individual root (or pair of roots) is involved. Posterior root involvement is usually bilateral. When roots at lower thoracic levels are implicated, the patient complains of pain round the girdles. The lightning pains of tabes are also a posterior root symptom. Root pains may also occur in herpes zoster.

There may be a sensory loss of radicular distribution. All modalities are affected but touch may be less affected than pain and temperature. Single root lesions may produce no sensory loss.

**Pain and paraesthesia in the arm (brachial neuritis)** The term brachial neuritis (brachial neuralgia) has been applied to all conditions characterized clinically by pain and paraesthesia in the upper limbs and/or shoulder girdle. Such common and predominant sensory symptoms in the upper limb can be produced by widely different conditions. Hence brachial neuritis is not a pathological entity.



**CAUSES** Some of the common conditions which can produce brachial neuritis are

(1) *Lesions of the cervical spine* (a) *Cervical disc prolapse* The pain is excruciating and of sudden onset. It is felt in the neck and down the arm and is frequently accompanied by stiffness and pain on movements of the neck. The pain is aggravated by coughing and sneezing. Paraesthesia may accompany the pain being present in the thumb and index finger (C5-6 root), middle finger (C7 root) or little finger (C8 root).

(b) *Cervical spondylosis* This condition is associated with degenerative changes in the cervical spine. The symptoms are subacute or acute in onset, the pain is of root distribution, and often extends down the entire arm and is referred to all the fingers. Paraesthesiae of a burning nature are often present.

Spinal cord tumours at the cervical level may occasionally be the cause of pain in the upper limb.

(2) *Thoracic inlet syndrome* Compression of the 8th C and 1st D roots by the enlarged transverse process or a small rib or fibrous band arising from the 7th C vertebra may result in pain along the inner border of the forearm and hand. Frequently, the subclavian artery is also pressed at the same site and the radial pulse on the affected side is usually feebler and can be obliterated by manoeuvres which tighten the scalenus anticus muscle.

(3) *Myofibrositis, periarthritis and arthritis of the shoulder* Pain and limitation of movements of the shoulder are complained of in addition to pain and paraesthesia if one of these causes is responsible.

(4) *Lesions of brachial plexus and of median and ulnar nerves* These may on occasion be the cause of sensory symptoms in the forearm and hand, but the characteristic distribution of sensory and motor disturbances render the anatomic diagnosis relatively easy, though the cause may not be obvious, an acute neuritis of unknown etiology may rarely be responsible. In the condition known as the carpal tunnel syndrome, the median nerve is compressed at the wrist by the transverse fascial ligament giving rise to paraesthesiae in the entire palm or in the distribution of the median nerve. These pains may radiate upwards to the neck and simulate a lesion situated at higher level.

(5) *Neuralgic amyotrophy* In this condition severe root pains occur and are followed by atrophy of muscles innervated by corresponding motor root. Muscles innervated by C5 are most commonly involved. History of vaccination, sera or fever prior to the onset may be present. Remissions and relapses may occur.

(6) *Cardiovascular conditions* The pain of coronary insufficiency may be felt on the inner aspect of the left arm and in the little finger. Occlusion of the brachial artery may occasionally be the cause of pain and paraesthesia.

Similar ischaemic mechanisms are responsible for the pain in polyarteritis nodosa, scleroderma and other conditions grouped under the term Raynaud's phenomenon

**Pain in the leg (Sciatica)** By the term sciatica is meant pain in the distribution of the sciatic nerve or its component nerve roots. The pain may be sharp or dull pain in the buttock, spreading down the posterior or lateral aspects of the thigh and leg to the dorsum or lateral aspect of the foot and usually aggravated by coughing, straining, sneezing or jolting of the spine. Sciatica may be due to a number of causes involving the spinal nerves in the spinal canal or in the intervertebral foramina, or the sciatic plexus in the pelvis. Dysaesthesiae and paraesthesiae may accompany the pain of sciatica.

**CAUSES** (1) *Root compression* *Herniated intervertebral disc* causing pressure on the nerve roots is the commonest cause of sciatica. Pain may be felt down the lateral aspect of the thigh and inner aspect of the leg up to the ankle (L4 root), or down the posterolateral aspect of the thigh and lateral aspect of the leg and across the dorsum of the foot (L5 root) or down the posterior aspect of the thigh and on the lateral aspect of the leg and foot (S1 root). Root pains are however not always felt in the whole distribution of the affected nerve.

Other causes of root compressions are vertebral disease, e.g. tuberculosis of spine and spinal tumour.

(2) *Pelvic conditions* Inflammation may spread to the nerve from the appendix, colon, or arthritis of the hip joint, or the nerve may be compressed by a neoplasm usually arising from the uterus.

(3) *Sciatic neuritis* Whether sciatic neuritis is a clinical entity is doubtful. Sciatic pain may occur in peripheral neuropathy complicating diabetes mellitus. Rarely a neurofibroma of the sciatic nerve may be responsible for the pain.

(4) *Spinal cord lesions* Vascular and other lesions of the spinal cord are rare causes of unilateral sciatica. The onset is acute and there is usually evidence of a rather extensive cord lesion. Spinal extradural abscess and arachnoiditis are other causes.

#### RULES OF EXAMINATION

Examination of the sensory system is the most difficult part of the whole neurological examination because it depends on the patient's interpretation of the stimuli administered, and because of the difficulties of standardizing sensory stimuli and gauging their response. Before beginning a sensory examination, the patient should be explained the exact nature of the test and carefully instructed as to what he is supposed to do. Care must be taken not to tire the patient out or prejudice his mind by suggestions. In order to ensure accuracy, the patient's eyes must be kept closed or covered. The patient is instructed to make his response prompt, as soon as the artificial stimulus is

perceived, by uttering the word "yes" to indicate his ability to feel the stimulus. Otherwise the phenomenon of delayed response, which indicates a delay of conduction, may be completely overlooked. The time elapsing between stimulus and response, under normal conditions, is one-tenth of a second; in disease states, it may be as long as 10 seconds. The patient under no circumstances must be asked "Do you feel that?", when a stimulus is applied. In comparing perception of sensation on corresponding parts of the two sides of the body, it may be difficult to decide whether the patient feels more sensation on one side or less sensation on the other. When results are inconsistent or confusing it may be necessary to examine the sensory system a number of times. In hysterical anaesthesia, in addition to a non-anatomical distribution of the sensory loss, it is common to find a change in the location of anaesthesia with successive tests.

The sensory functions are examined, with a view to determining whether they are abnormally active, absent or perverted. It is frequently possible to locate the exact seat of trouble through a sensory examination, because the pattern and quality of sensory loss for each type of lesion, whether of the peripheral nerve, sensory root, spinal cord, thalamus, sensory cortex or parietal cortex, are different.

The results of each sensory examination are accurately mapped out on the skin with a skin pencil, and then transferred to charts meant for recording sensory changes (Fig. 13.85). Such charts are useful not only for determining whether the cutaneous area corresponds to a radicular zone, peripheral nerve region or cord segment, but also for determining whether a neurological lesion is progressive, stationary, or regressive by comparing with charts obtained through subsequent tests.

For clinical purposes, the best way of carrying out a rapid sensory examination is to do a rapid survey of responses to light touch, pin-prick, vibration and joint sensations and deep pain. If any abnormalities are found, a more careful clinical assessment of the sensory changes can be carried out.

In stuporous or semi-conscious patients response to painful stimuli in the form of wincing, facial grimacing or limb withdrawal may be the only means available of assessing sensory function.

#### TYPES OF SENSATIONS

General sensation may be classified into two main types, exteroceptive and proprioceptive.

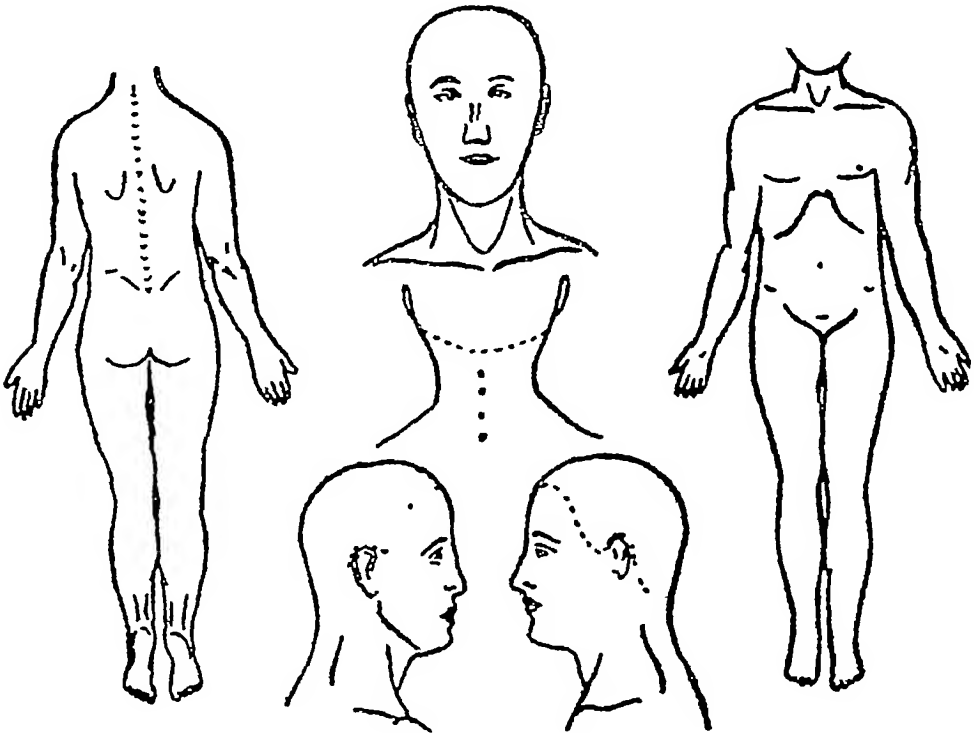
##### Exteroceptive sensations.

**TOUCH OR TACTILE SENSATIONS** are tested with a wisp of cotton wool or a fine, soft paint brush touched lightly to the skin, the patient being instructed to say "yes" immediately on feeling the touch. It is customary to start with the face and go down the body, comparing symmetrical areas on either

side Light pressure perception may be tested with the head of a pin Touch sensation is recorded as anaesthesia when absent, hypoaesthesia when reduced, and hyperaesthesia when excessive Whilst testing for touch, it should be borne in mind that some areas of the skin are tougher and less sensitive than

RECORD NO \_\_\_\_\_

NAME OF PATIENT \_\_\_\_\_ AGE \_\_\_\_\_ DATE \_\_\_\_\_



Sig \_\_\_\_\_

- 1) Increasing severity of sensory impairment to be shown by closer marking
- 2) Separate charts for different sensory modalities, if they do not coincide

- - - = Loss or Impairment to Light Touch  
 // // // = Loss or Impairment to Pin Prick  
 O O O = Loss or Impairment to Heat & Cold  
 . . . = Loss or Impairment to all Superficial modalities

Fig 13 86 Chart for recording sensory changes

others Hairy surfaces over the body can conduct touch sensations through the presence of special receptors in the hair roots

The *power of localization* is investigated by touching the skin with a pencil or finger tip with the patient's eyes closed, and asking him to put a finger on the same point Any error of more than 2 inches is usually considered abnormal, it depends however on the area of the body investigated

PAIN or *superficial algnesia* is usually tested with a pinprick, the prick being hard enough to elicit an unpleasant sensation To control the degree of pin prick, it is advisable to hold the pin in such a way that its point just extends beyond the tip of the examiner's middle finger An area of superficial hyperaesthesia (hyperalgnesia) is usually easily detected by stroking the skin with a pin The moment the hyperaesthetic area is touched, the patient winces An area of diminished sensation can also be similarly tested, by stroking the skin with a pin, preferably from the anaesthetic zone outwards In order to avoid summation of stimuli, which may prove deceptive, similar strokes with a pin are tried out in other parts of the body for comparison

*Hyperpathia* is a term used to describe a condition where there is an exaggerated response to painful stimulus, tending to make the stimulus intensely unpleasant The threshold for pain may also be raised

TEMPERATURE Temperature sensation is conducted by fibres that anatomically closely follow those of pain It is tested with the aid of two large test tubes, preferably made of copper or steel but not glass, and filled with warm (hot water stimulates pain fibres), and cold water (or ice) respectively The patient must not be asked to say whether the stimulus is hot or cold but what type of feeling he gets For accurate comparison, only symmetrical areas of skin should be tested, because the uncovered parts of the body, such as the distal parts of the limbs, are less sensitive to cold than the covered parts Loss or diminution of temperature sense, as well as over-reaction to temperature stimuli, should be noted Inability to differentiate hot and cold, or confusion of one with the other, is not uncommon in syringomyelia

Minor differences of temperature ( $2^{\circ}$  to  $5^{\circ}$ ) of objects, such as test tubes filled with warm water at different temperatures, may also at times be tested

*Dissociated Anaesthesia* Loss of pain and temperature sense with preservation of touch and other forms of sensibility is called *dissociated anaesthesia* Dissociated anaesthesia is always due to a lesion situated below the pons because it is here that the lateral spinothalamic tract joins the medial lemniscus The following are the causes of dissociated anaesthesia (1) A lesion situated in the centre of the spinal cord causing bilateral and segmental loss of pain and temperature sensation with retention of touch The common causes are syringomyelia, intramedullary tumour and haematomyelia (2) Hemisection of the cord giving rise to contralateral dissociated anaesthesia in the lower limb

and sometimes the trunk (3) Anterior spinal artery thrombosis The anaesthesia may be transient (4) Lesions involving the lateral medulla, for example thrombosis of posterior inferior cerebellar artery and syringobulbia

**RELATIONSHIP OF SENSORY LEVEL TO SPINOUS PROCESS** In order to accurately localize the level of a lesion in case of sensory loss, it is necessary to bear in mind that the different spinal segments are not situated at the same levels as their numerically corresponding vertebrae This is because the lower end of the spinal cord in an embryo extends to the lower end of the spinal canal, but ascends upwards as growth proceeds, pulling the spinal roots of various segments to higher levels In an adult the lower end of the cord corresponds to the level of the first or second lumbar vertebra

The relationship between the segments of the cord and the vertebral column can be summarised thus Between C1-C4 the relationship is direct From C5-C8 subtract 1, e.g. for C8 cord segment, the corresponding vertebra is C7 From T1-T9 subtract 2, and between T10-S5 subtract 3 to obtain the corresponding vertebral segment

The anatomical relationships of various spinal cord segments to corresponding spinous processes (in brackets) in an adult are as follows

C 1 - 2 (C 1)	T 2 (T 1)	T 10 (T 8)
C 3 (C 1)	T 3 (R 2)	T 11 (T 9)
C 4 (C 2)	T 4 (T 2, 3)	T 12 (T 10)
C 5 (C 3)	T 5 (T 3, 4)	L 1 (T 10, 11)
C 6 (C 4)	T 6 (T 4, 5)	L 2 (T 11)
C 7 (C 5)	T 7 (T 5, 6)	L 3 (T 11, 12)
C 8 (C 6)	T 8 (T 6)	L 4, 5, S 1 (T 12)
T 1 (C 7)	T 9 (T 7)	S 2 - 5 (L 1)

### Proprioceptive sensations.

**JOINT SENSE** The patient is asked to say "yes", each time he feels movement at a particular joint, when slow and passive movements are being carried out by the examiner He is then asked to say in which direction the movement had taken place Each joint is examined individually The big-toe is usually selected for testing The examiner should hold the sides of the distal phalanx with the right hand, whilst the left hand is used to steady the interphalangeal joint The phalanx is moved up and down taking care to avoid a push-pull stimulus The patient must understand, however, what is exactly meant by "up" or "down" movement of the toe He is asked to shut his eyes during the test and state the direction of movement of the toe in relation to its neutral position To begin with, the test movements have to be somewhat large, once the idea of the test becomes clear to the patient, the smallest possible movements that are detectable should be employed, avoiding sudden or quick jerks at all times Joint sense (along with vibration sense) is a useful indicator of integrity or disease of the posterior column or roots

**SENSE OF POSITION AND PASSIVE MOVEMENT** After taking hold of any one of the patient's extremities, the examiner moves it in various directions, the blind-folded patient being asked to imitate these motions with the corresponding limb of the opposite side. Alternatively, the examiner may place one limb in a certain position and ask the patient to describe the exact position in which it is placed. For all these tests, the patient must keep his limbs completely flaccid, avoiding all forms of voluntary movement. The examiner must exert uniform pressure throughout the range of movement so as to give the patient no information about the movement through superficial sensations.

Interference with postural sensibility may be due to a lesion involving any part of the sensory motor cortex.

**DEEP PRESSURE PAIN** Deep pain can be elicited by either applying strong pressure or by pinching the muscles of the forearm, thigh or calf or the tendo-Achilles. Testicular pressure may also be tried. Deep pressure pain tends to be diminished or absent in tabes and tabo-paresis, and is frequently exaggerated in neuritis. When deep pain is diminished but not lost, the response may be delayed.

The *nerve trunks* should also be tested by pressure or gentle percussion. In case of peripheral nerve injuries, if the regenerating neuraxes have reached the point of pressure, percussion or pressure on the nerve will elicit a tingling sensation (Tinel's sign).

**Thickening of nerves** In the initial stage of tuberculoid leprosy, neurologic manifestations may precede skin lesions and there may be thickening of nerves along their superficial course. The ulnar nerve in the elbow, the peroneal nerve in the popliteal fossa and the major auricular nerve crossing the sternomastoid may be palpable (Fig. 13.87).

**VIBRATION SENSE** The vibration sense is of particular value in the diagnosis of disease of the posterior columns. The sensation of vibration must first be demonstrated to the patient by pressing the base of the tuning fork (128 cps) on the sternum, both during and after cessation of vibration. The patient is asked to say "buzzing" or "yes", when he feels the vibration, and "no" when he feels only pressure. When this is made clear to the patient, he is asked to close his eyes and the vibration sense tested. The bony points convenient for the test are the malleoli, the tibial tuberosities, the anterior superior iliac spines, the radial and ulnar tuberosities and the points of the elbows. The patient must also indicate when he stops feeling the vibration, so that this can be compared with the examiner's own perception. When the vibrating fork is no longer felt on one bony point, but can still be felt buzzing on a comparable point on the other side of the body, the sign is significant of disease, especially if the difference on the two sides be consistent.

Since the sensation of vibration is identified at thalamic level, whilst that of passive movement or sense of movement requires cortical participation also

for its recognition, a significant disturbance in the identification of passive movement with preservation of vibration sense suggests a lesion above the thalamus. Impairment or loss of perception of vibration is suggestive of disease of posterior columns, roots, peripheral nerve or parietal lobe. Vibration sense is normally impaired in old age.

The appreciation of vibrations of a tuning fork may be diminished or lost even when all other forms of sensibility are intact. This is a particularly sensitive index of posterior root and posterior column lesions.

## CORTICAL SENSORY FUNCTION

1 *Stereognosis* Normally, a person with eyes closed can recognize an object placed in the hand by its feel. This is dependent mainly on the sense of position and of movement but superficial sensibility also plays some part, hence the term "combined sensibility" for the sense of stereognosis. The patient is asked to close his eyes, various familiar objects (such as coins and keys) are placed in each hand, and he is asked to name each. If he is unable to name them, he may be asked to describe each in terms of size, shape and texture. Do not use a bunch of keys, since the sound of rattling may betray its nature to the patient. The patient may either fail to recognize an object (astereognosis), or may hesitate in his answer (dysstereognosis), the time should therefore be recorded for each answer. The test should be carried out simultaneously with both hands, using identical objects.

It is worth noting that a hemiplegic hand, which remains astereognostic, is practically useless even if it regains its motor power completely.

When the elementary sensory qualities are completely lost or show severe blunting, astereognosis is invariably present (secondary astereognosis), failing this, astereognosis should be considered an independent and important sign of a contralateral parietal lobe lesion. Although, most commonly encountered in lesions of the post-central gyrus, astereognosis may occur in lesions involving the subcortical parietal region, thalamus or the lower part of the medulla.

2 *Two point discrimination* This is tested by simultaneously touching areas of the skin with the two points of an ordinary compass or hair pin or the heads of two ordinary pins. The patient's eyes being closed, the points are placed on the skin, sufficiently wide apart to be recognized instantaneously as two points (double contact) and then gradually brought together, until the patient reports being touched with a single point only. The minimum distance between the two points that is appreciated as a double contact by the patient is noted. Convenient skin areas for testing tactile discrimination or two-point distinction are the palms and soles (where the patient can normally distinguish points 1.5 to 2 cm apart), dorsal surfaces of hands and feet (3 cm), the finger tips (3-8 mm) and the shins of the legs (4 cm).



Disturbances in tactile discrimination may be met with in lesions involving any part of the sensory motor cortex and posterior columns but are most conspicuous in parietal cortical lesions. They can also be seen in peripheral nerve and root lesions.

3 *Sensory extinction* When two stimuli, with cotton wool, are simultaneously applied to two identical points on either side of the body, a patient with tactile extinction will fail to appreciate individual touch on the affected side, a sign of diagnostic value in parietal lobe lesions.

4 *Figure identification* (Graphaesthesia) When figures or numbers are written with a match or blunt point on the skin surface of a patient with eyes closed, the patient is usually able to recognize these provided they are of sufficient size, not written too rapidly and varying from region to region.

5 *Weight sense* (Barognosis) This is tested by using two coins of different weight but of equal or nearly equal size. They are placed alternately and synchronously on the extremity or the part to be tested, the patient being asked to indicate which is heavier. The capacity to detect weight differences is less on the affected side. When objects of equal weight are placed in both hands, the one in the affected hand always feels lighter in weight. The cerebral centre for weight perception is said to be in the parietal area.

6 *Localization of touch* (Topognosis) This consists of ability to localize stimuli applied to parts of the body with the eyes shut.

#### Common patterns of sensory abnormality

*Complete loss of all sensation below a clear cut level* occurs in a total transverse lesion of the cord (Fig. 13 88a). There may be a zone of hyperaesthesia at the upper level of the lesion.

*Characteristic distribution of cutaneous analgesia* is found in tabes dorsalis (Fig. 13 88b). The segmental regions involved include the lower cervical and upper thoracic segments, and there is also analgesia round the tip of the nose and the lips.

*Dissociated anaesthesia* (Fig. 13 88c). Impairment of pain and temperature sensation over several segments with normal sensation above and below is suggestive of a lesion situated in the centre of the cord thus involving the crossing fibres. It is called a suspended sensory loss because it has an upper as well as a lower level.

*Unilateral loss of pain and temperature sensation below a definite level* (Fig. 13 88d). A unilateral lesion of the spinal cord above the level of the 12th thoracic segment causes loss of pain and temperature sensation on the side opposite the lesion, loss of postural and vibration sensations on the side of the lesion. On the side of the lesion at the highest level there is a band of analgesia as a result of involvement of the root entry zone.

*Loss of pain and temperature on one side of the face and contralateral side of the body (Fig 13 88e) Thrombosis of the posterior inferior cerebellar artery affects the descending root of the trigeminal nerve and the ascending spinothalamic tract from the rest of the body*

*Unilateral loss of all forms of sensation (Fig 13 88f) Total hemianalgesia is found with thalamic or upper brain stem lesion*

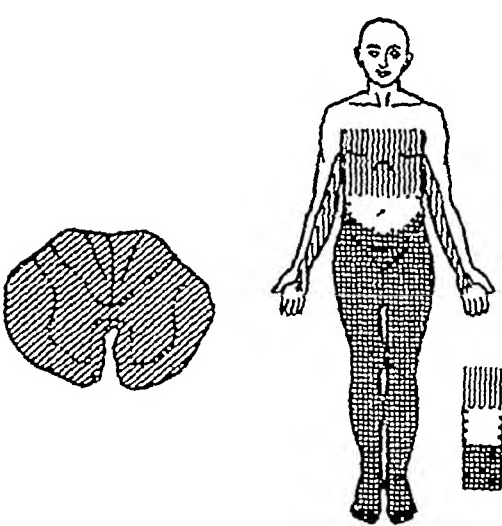


Fig 13 88 a Transverse lesion of the cord.

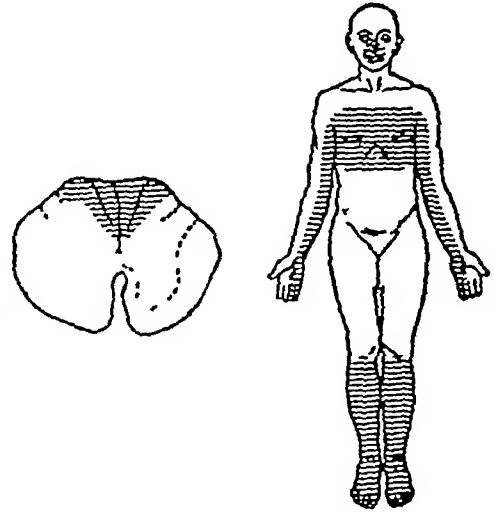


Fig 13 88 b Sensory loss in tabes dorsalis

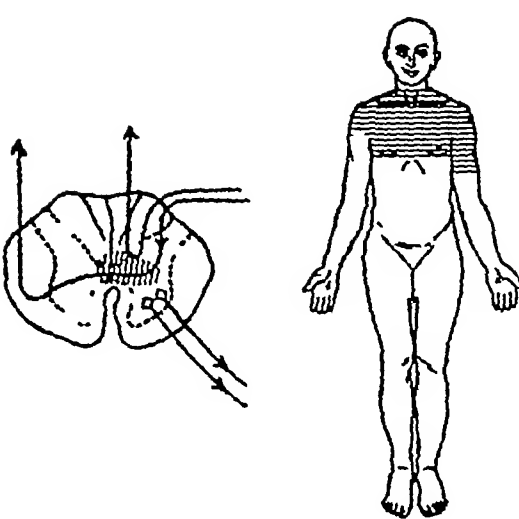


Fig 13 88 c Dissociated anaesthesia  
Central cord lesion

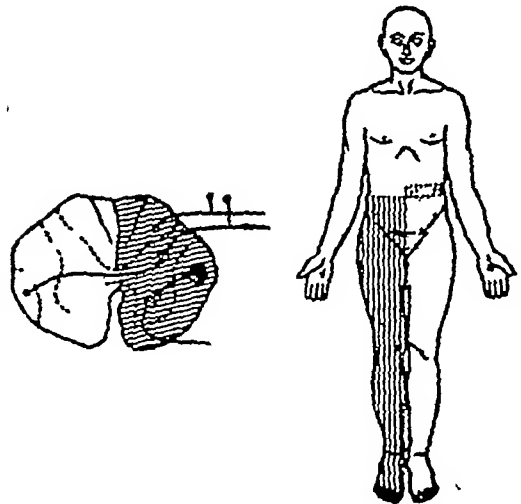


Fig 13 88 d Unilateral loss of pain and  
temperature in hemisection of the cord

*Loss of all forms of sensation over sacral segments (Fig 13.88g) A saddle analgesia indicates a cauda equina or conus medullaris lesion*

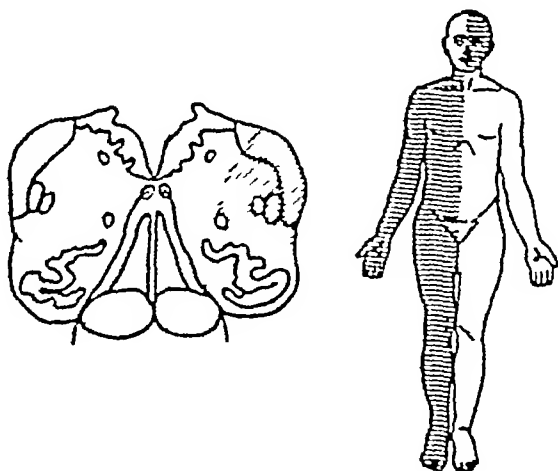


Fig 13.88 e Posterior inferior cerebellar artery lesion

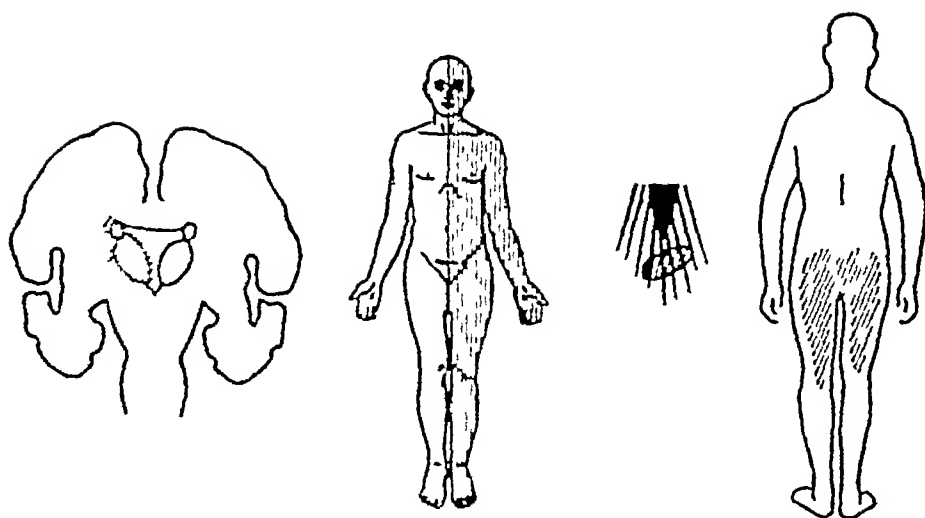


Fig 13.88 f Total hemi-analgesia in upper brain stem or thalamic lesion

Fig 13.88 g Saddle analgesia in cauda equina or conus medullaris lesion

## REFLEXES

Reflexes may be defined as involuntary motor responses to the sensory stimulation of stretch receptors in tendons and muscles. The "taking" or eliciting of reflexes is perhaps the most important part of a neurological examination. It affords valuable information about the diagnosis and localization of neurological lesions, and does not depend on the cooperation and intelligence of the patient, as in the case of motor and sensory examinations. The taking and interpretation of reflexes requires skill and practice.

**Reflex arc (Fig 13 89)** The fundamental nature of the reflex arc is the same in all reflexes, consisting of (1) a receptor at the periphery, (2) an afferent or sensory neurone, for carrying the impulse to a motor cell in the spinal cord or medulla, (3) an efferent or motor neurone, for delivering the stimulus peripherally, and (4) the effector in the muscle or gland, which produces the response.

Interruption of the reflex arc at any point abolishes the response.

### TYPES OF REFLEXES

Three main types of reflexes are recognized: (1) superficial, (2) deep, and (3) visceral.

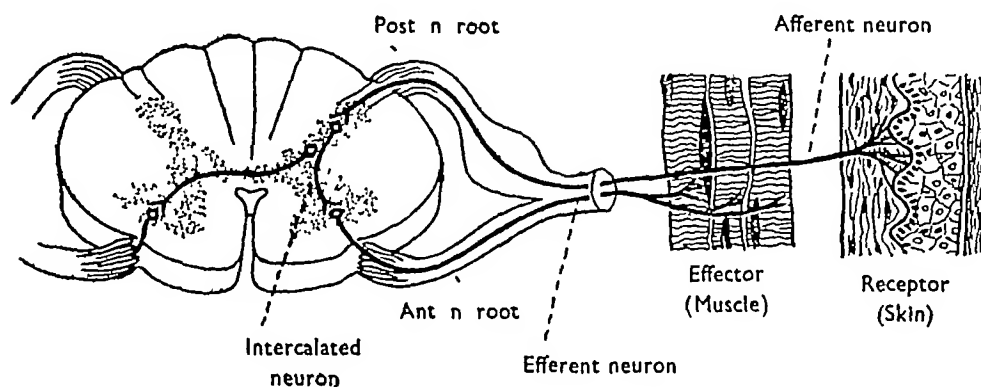


Fig 13 89 Diagram of reflex arc

### SUPERFICIAL REFLEXES

The superficial reflexes are usually elicited by the stimulation of certain parts of the skin, mucous membrane or cornea, the end result being the contraction of one or more muscles. The localizing value of superficial reflexes is limited, because the level at which they are lost may be far below the level of the causative lesion. They may be abolished even by lesions in the cerebral hemispheres.

**CORNEAL REFLEX** (Cranial Nerves V and VII) A wisp of cotton wool is used for touching the cornea, the eye being approached from the side to avoid a winking reflex (Fig 13 90) The normal response consists of an instantaneous closure of the eyes, the reflex being mediated through the fifth (afferent) and seventh (efferent) cranial nerves

A loss of the corneal reflex is often an early and important sign of a trigeminal nerve affection or cerebellopontine angle tumour

**CONJUNCTIVAL REFLEX** (Cranial Nerves V, VII) Reflex contraction of the orbicularis palpebrum muscle as the result of touching the conjunctival mucosa with a wisp of cotton wool

**PHARYNGEAL REFLEX** (Cranial Nerves IX, X) A superficial reflex elicited by touching or tickling the mucous membrane of the pharynx, resulting in "gagging"

**PALATAL REFLEX** (Cranial Nerves IX, X) A superficial reflex elicited by touching the soft palate, resulting in an elevation of the arch of the soft palate and uvula.

**ABDOMINAL REFLEXES** (Epigastric T 6-T 9, Mid-abdominal T 9-T 11, Hypogastric T 11-L 1) The abdominal reflex is a skin-muscle reflex Here, on stimulation of the skin, the abdominal muscles contract. The abdominal reflexes probably utilize a short spinal reflex arc The centripetal impulses enter the lower thoracic spinal segments by way of the corresponding posterior roots, and its centrifugal impulses leave the lower thoracic segments by way of the corresponding anterior roots The impulses subserving these reflexes seem to utilize an elaborate arc in which there are several connector neurones The afferent supraspinal pathways pass up the cord, presumably to the region of the motor cortex, and efferents pass down into the spinal cord in or near the pyramidal tracts The existence of this anatomical pathway explains why the abdominal skin reflexes are abolished not only by lesions of the lower thoracic segments, but also by pyramidal lesions at much higher levels The details about these pathways are not known However, if local and segmental lesions are excluded, abnormality of abdominal reflexes point to affection of corticospinal tracts

Proper relaxation of the abdominal wall must be obtained by making the patient lie on his back with the knees comfortably drawn up Stroking the abdominal wall with a pin or point of a pencil or a pinwheel well to the side of the midline will elicit a contraction of the abdominal muscles on the same side The strokes are usually made at three levels, epigastric, mid-abdominal and hypogastric, on each side, and should preferably be directed towards the umbilicus The response is mainly segmental, being maximal at the level of the stimulus In the mid-abdominal area, one should look for a deviation of the umbilicus, which can be seen more easily than muscle contraction.

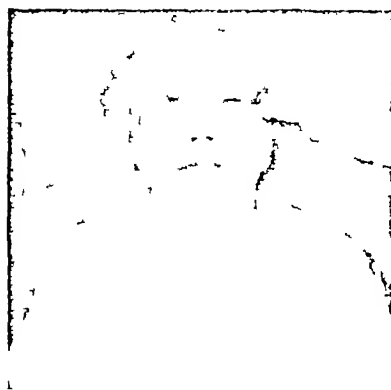


Fig 13 90 Method of eliciting  
corneal reflex The reflex is  
absent in this case



Fig 13 91 Method of eliciting  
plantar reflex

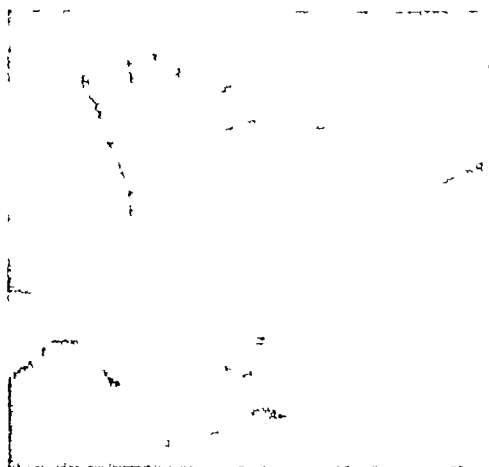


Fig 13 92 Extensor plantar response



Fig 13 95 Perforating ulcer of  
sole of foot in tabes dorsalis

Fig. 13 93, see p 516  
Fig 13 94, see p 519



Abdominal reflexes may not be elicitable in cases of obese, distended or flaccid abdomens as after repeated pregnancies, and in elderly subjects. Asymmetry is obviously significant.

Loss of abdominal reflexes, if unilateral, suggests pyramidal lesion, provided local and segmental lesions are excluded. The loss is not always proportional to the severity of the pyramidal lesion. In disseminated sclerosis, the abdominal reflexes may be lost early in the course of the disease. Easily fatigued abdominal reflexes in the young may be a sign of early pyramidal disease.

The abdominal reflexes may be preserved in the presence of other obvious evidences of "pyramidal tract lesions" as in some cases of cerebral palsy, amyotrophic lateral sclerosis, lathyrism. In cases of "acute paraplegia" and "hemiplegia" abdominal reflexes which are lost initially may reappear later.

**CREMASTERIC REFLEX (L 1, L 2)** Stimulation of the inner surface of the thigh in man causes reflex contraction of the cremasteric muscle, with elevation of the testicle on the same side. The reflex is absent in pyramidal tract lesions and in lesions affecting the reflex are such as vasectomy or varicocele.

**GLUTEAL OR ANAL REFLEX (S 4, S 5)** When the skin around the anus is scratched or pricked, a reflex contraction of the external sphincter of the anus is observed.

**PLANTAR REFLEX (L 5, S 1, S 2)** Stimulation of the sole of the foot, from the heel towards the toes, normally causes a plantar flexion of the toes. The toes must be relaxed for the test, if the patient is not sufficiently relaxed, he should be engaged in conversation or his attention distracted. When the feet are cold, the plantar reflex is weak or absent. The stimulus, which should be gentle at first (a blunt object such as the point of a key) should begin near the heel, be carried upward and laterally along the sole until the ball of the foot is reached, and then inward, across to the medial side without touching the toes (Fig 13 91). Usually a pin or a pencil is used. The stimulus may start at the midpoint of the outer border of the sole and is then carried downwards towards the heel. The stimulus need not be painful, unless a reflex response is not obtained by ordinary stimulation. A sudden or vigorous stimulation may cause a forceful withdrawal of the leg. When the response to the stimulation of the sole consists of dorsiflexion of the great toe and fanning out of the other toes, it is called extensor plantar response or positive Babinski's sign (Fig 13 92).

*Significance of extensor plantar response* While the method of eliciting the plantar response is simple, the response obtained is often confusing and at times variable. Extensor plantar response is an important sign and perhaps the most famous sign in neurology. The variability of the response and the practical difficulty in differentiating a pathological extensor response from a



normal withdrawal to a stimulus, should temper one's attitude to the unfortunately much publicised reliability of this sign in the diagnosis of pyramidal tract lesion. The extensor plantar response is a physical sign that is correlated with other clinical features. The following points are of relevance.

(1) Electromyographic studies have shown that stimulation of the border of sole results in contraction of two groups of muscles — (a) *hallucis brevis* and foot dorsiflexors (b) Extensors digitorum, extensor hallucis, tensor fascia lata and semitendinosus. Normally, contraction of the first group is stronger producing flexor response. In pathological conditions producing extensor response there is recruitment of second group of muscles and even though the "flexors" contract they are overpowered by "extensors".

(2) The abnormal response consists of (a) dorsiflexion of the big toe (b) abduction and fanning of other toes. There is much confusion as to whether the term extensor plantar response could be used when only one component is present. The majority seem to accept the dorsiflexion of big toe as being more important.

(3) Several studies (experimental, clinical and clinicopathological) have shown that (a) an extensor response may be present when there is no lesion of the corticospinal tract (b) Cases proven at autopsy to have damaged corticospinal tracts have had normal plantar response (c) In cases of hemiplegic, diplegic and tetraplegic forms of cerebral palsy where the expected findings suggestive of pyramidal tract lesion are absent, the plantar response may be flexor even in presence of brisk reflexes, clonus and spasticity (d) In cases of athetosis or dyskinesic syndromes often 'spontaneously' show extensor response though direct stimulation may produce flexor response (e) A transient extensor response may be obtained in sleep after a long march and general anaesthesia.

The plantar response, therefore, must be evaluated with the other signs. A consistent extensor response on several examinations from the same patient is probably of significance. It is possible that tracts other than corticospinal tracts are also concerned in the supraspinal influence on the plantar response.

There are many pyramidal tract responses in the lower extremities which are characterised by dorsiflexion of the toes. Some of these are important in that they can be elicited where for some reason the plantar surface of the foot cannot be stimulated. The modifications are as follows:

*Gordon's leg sign* A Babinski-like response on squeezing the calf muscles.

*Oppenheim's sign* Firm pressure downwards with thumb and index finger, along the medial surface of the upper third of the tibia, to elicit a Babinski-like response.

*Chaddock's sign* A response similar to that of Babinski's sign on stroking the lateral malleolus.

*Gonda's reflex* Upward movement of the big toe, produced by pressing one of the other toes downward and then releasing it with a snap

*Stransky reflex* A slow but vigorous adduction of the little toe followed by its sudden release results in a dorsiflexion of the great toe

*Schaefer's sign* Babinski-like reaction on squeezing the Achilles tendon

*Rossolimo's sign* Flexion of the toes, on quick percussion of the tips of the patient's toes with the finger tips

*Mendel-Bechtrew sign* Flexor movement of four outer toes induced by tapping the dorsum of the foot in the region of the cuboid bone

## DEEP REFLEXES

A deep reflex (*tendon reflex* or muscle stretch reflex) is usually elicited by delivering a sharp tap or blow on a tendon or bony prominence in order usually to cause sudden stretching of a muscle or tendon. The tap, by stretching the tendon momentarily, stimulates sensory end organs or receptors, which in turn starts the reflex action. In general, in the absence of lesions involving any of the components of the reflex arc, the activity of muscle stretch reflexes indicates the balance between excitatory and inhibitory impulses, reaching the lower motor neurones from various higher centres.

**TECHNIQUE OF ELICITATION** There are several methods of eliciting deep reflexes, but certain requirements are common to all these methods. (1) The patient must keep the limbs completely relaxed. He must be asked not to help the examiner in any way, in short just do nothing. (2) An optimal amount of tension should be produced in the muscle, by stretching it through passive movement and by favourable positioning of the extremity. (3) The method of application of the stretch stimulus in case of a deep reflex usually consists of a sudden sharp blow, struck with a percussion hammer. Whenever necessary, successive blows may be struck with different degrees of severity in order to judge the degree of response. (4) Certain reflexes such as the biceps jerk, are best elicited by indirect percussion, that is by placing the examiner's finger or thumb over the tendon of the muscle before delivering the stroke.

**REINFORCEMENT OF REFLEXES** (Fig 13 93) When a reflex is difficult to elicit in spite of relaxation, efforts should be made to reinforce the reflex, through some determined voluntary act, with a view to increasing muscle tone throughout the body. The patient may be asked to reinforce by (a) hooking the fingers of both hands and then exerting a strong pull, (Jendrassik's method) (b) By clenching the teeth. (c) Another effective method of reinforcement is to induce a mild voluntary contraction of the muscle that is being tested eg active plantar flexor of the foot. The contraction must be sufficient enough to tighten the muscle but not to produce movement of the joint, lest the reflex be abolished altogether by a powerful contraction of the muscle. (d) Forcibly raising the head.

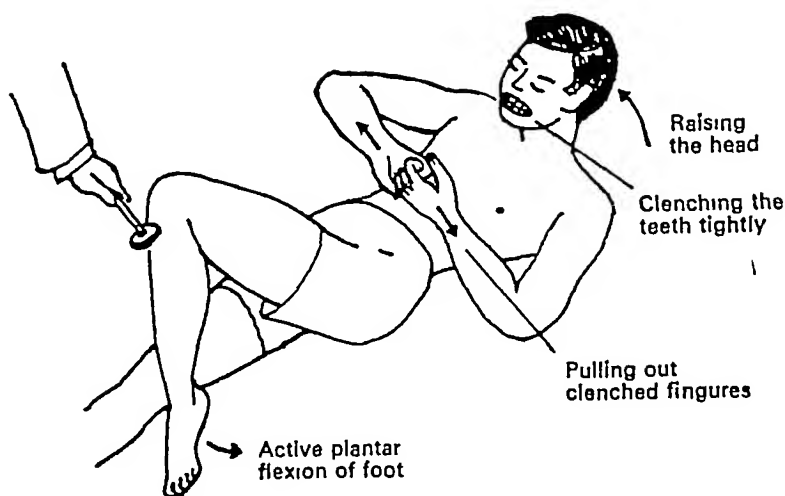


Fig 13.93 Methods of reinforcement of deep reflexes

**GRADING OF RESPONSES** An assessment of the activity of tendon reflex demands consideration of several features—the speed, force or amplitude and duration of contraction, and the rapidity with which it is checked. Some method of grading of reflexes is therefore desirable in clinical practice. The following alternative methods of recording are therefore recommended.

0 Absent		0 Absent
1 Sluggish	or	(+) Sluggish
2 Active		+ Present
3 Very active	or	+ (+) Brisk
4 Clonus		+ + Exaggerated
		+ + + Clonus

**Important reflexes** Although almost every muscle of the body is capable of contracting reflexly to a stimulus capable of stretching it, it is customary to study the following deep reflexes only during routine neurological examination.

**JAW JERK (Mid-pons)** The mouth is kept slightly open and the examiner's finger placed firmly over the chin, the finger is then struck with a small hammer or with another finger. Alternatively, a pencil may be laid on the lower teeth and tapped. Sudden closure of the jaw through muscle contraction is observed. Normally the jerk is very sluggish and may even be absent. Marked exaggeration of the jaw jerk suggests an upper motor neurone lesion above the level of the pons and may occur in pseudobulbar palsy, motor neurone disease and disseminated sclerosis.

**PECTORALIS REFLEX (C5, C6, C7, T1)** The patient adducts the arm slightly and the tendon of the pectoral muscle, near its insertion on the humerus, is struck either directly or (preferably) indirectly with the examiner's left hand interposed. Contraction of the muscle can be both seen and felt.

**BICEPS REFLEX (C 5, C 6)** With the patient's arm semiflexed at the elbow and resting on the thigh or on the examiner's forearm, the examiner after placing his thumb over the patient's biceps tendon strikes it with a hammer. The forearm will be seen to flex with a jerk.

**BRACHIORADIAL (Radial periosteal) REFLEX (C 5, C 6)** On striking the styloid process of the radius in the semipronated forearm, there is contraction of the brachioradialis and flexion of the elbow. The biceps often contracts as well and there may be slight flexion of the fingers. The reflex is said to be "inverted" if finger flexion is the only response. This is associated with an absent biceps jerk. It is probably explained by loss of spinal reflex in part of its course through the spinal cord in segments C 5, C 6 associated with a lesion of pyramidal tract at the same level. The latter causes a widening of the zone of provocation of reflexes below the level of the lesion. The muscles which flex the fingers and thumb are innervated at a lower level than C 5, C 6 and thus show a brisk stretch reaction. At the same time the triceps jerk (C 7, C 8) is also brisk. An inverted supinator reflex indicates a cord lesion at the level of the fifth cervical segment causing a lower motor neurone lesion of C 5 and an upper motor neurone lesion of reflexes innervated below this level. It is fairly common in cervical disc lesion, syringomyelia, trauma to the cervical region and at times in cervical neoplasms.

**TRICEPS REFLEX (C 6, C 7, C 8)** The patient is made to flex the semipronated arm, which is pulled across the chest, and the triceps tendon struck just above the olecranon process to elicit a contraction of the triceps muscle.

**Inverted triceps reflex** If on tapping the olecranon, the response consists of flexion of the forearm without any contraction of the triceps, it is called inverted triceps reflex. It signifies a cord lesion at C 8 level.

**FINGER FLEXION REFLEX (C 7, T 1)** The patient partially flexes the terminal phalanges of his fingers. The examiner then places his middle and index fingers on the palmar surfaces of the phalanges and strikes them with a hammer. Flexion of the fingers will be seen and felt.

**KNEE JERK (Quadriceps reflex, patellar reflex) (L 2, L 3, L 4)** This reflex can be elicited in one of several ways. (a) *Sitting position* The patient is made to sit on the edge of a high bed, couch or chair, with the legs dangling freely over the edge. The patient may then cross one knee over the other, or alternatively, the examiner slides his own wrist underneath the knee to be tested, to afford support. The patient's attention is then diverted by engaging him in conversation and the patellar tendon struck sharply with a hammer half-way between the patella and the insertion of the tendon in the tibia. (If a proper hammer is not available, the ulnar border of the examiner's hand or the edge of the diaphragmatic chestpiece of a stethoscope may be used to deliver the blow.) The leg will be seen to move or jerk forward with the contraction of the quadriceps muscles. In case the patient's feet are planted on the floor,

the legs may be moved forward with toes slightly plantarflexed and the tendon then tapped (b) *Supine position* In the supine position, the examiner places his hand or forearm underneath the knee to raise it off the bed, and then strikes the patellar tendon with a hammer Alternatively, he may semiflex the leg and thigh to an angle of  $20^\circ$ , rotate the extremity outward, and holding it in this position by grasping it beneath the knee, he strikes the blow Yet another method of eliciting the reflex is to push the patella downwards with the index finger, in order to stretch the quadriceps muscle, and then tap the finger with a hammer, in a downward direction Reflex contraction of the muscle pulls the patella up The method is useful when the lower leg has been amputated

**ANKLE JERK** (Gastrocnemius and soleus reflex) (L 5, S 1, S 2) By tapping the tendo-Achilles, the gastrocnemius-soleus group of muscles contract and plantarflex the foot (a) *Sitting position* The patient sits with his legs dangling, upward pressure is exerted on the ball of the foot to secure proper tension and the tendon tapped briskly with a hammer Alternatively the patient kneels on a stool or chair, with his feet hanging over the edge The ankle is then dorsiflexed sufficiently to obtain optimal tension of the tendon (or the foot grasped and passively dorsiflexed) and then the blow delivered (b) *Supine position* Partially flex the hip and the knee, rotate the extremity outwards, grasp the ball of the foot, dorsiflex the foot so as to tighten the tendo-Achilles and then tap the tendon briskly with varying degrees of strength (c) *Prone position* The patient is made to lie on his face, the hip is extended and the knee flexed at right angles The ankle is slightly dorsiflexed so as to put the tendon on moderate tension A sharp tap is then given on the tendon

**Significance of reflexes** Normally, all superficial and deep reflexes are elicitable and equal on the two sides Inequality of reflexes, either superficial or deep, on the two sides (or a discrepancy between the two) is suggestive of pathological disturbance of the nervous system When one or more pathological reflexes are also demonstrable anywhere in the body, the presence of organic nervous disease becomes doubly certain

**SUPERFICIAL REFLEXES** The significance of these has already been discussed

**DEEP REFLEXES** *Exaggeration of deep reflexes* Since the deep reflexes are normally under partial inhibition by the higher centres, lesions of the motor cortex or pyramidal tracts (upper motor neurone) result in hyperactive or exaggerated deep reflexes Hyperactive reflexes may also be observed in tense individuals, in functional disorders, anxiety states and strychnine poisoning

*Diminution or absence of deep reflexes* 'This may result from any lesion which interrupts the reflex arc (Fig 13 94) The lesion may be in the sensory

or motor root, anterior horn, peripheral nerve or peripheral muscle myxoedema, there is a characteristic slowing of the relaxation of the tendon reflexes (Wotman's sign). A delayed relaxation of the ankle jerk may also be observed in diabetes mellitus, hypothermia, obesity, gross oedema, and use of drugs such as propranolol, reserpine or quinidine, Parkinsonism, pernicious anaemia, neurosyphilis, sarcoidosis, hypokalaemia, and myasthenia gravis.

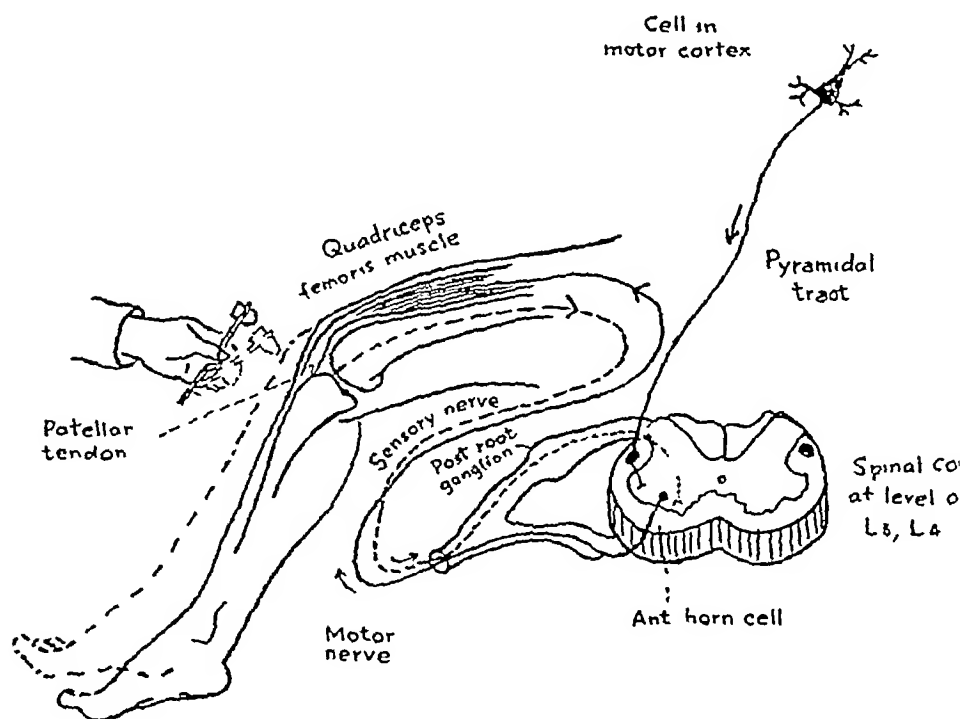


Fig 13.94 Diagram of the reflex pathway of the knee jerk. Destruction of the pyramidal tract causes hyper-reflexia. Destruction of the lower motor neurone causes hyporeflexia or areflexia. Such a lesion may be in the (i) anterior horn cell, it may be acute (e.g. anterior poliomyelitis) or chronic (e.g. motor neurone disease), (ii) anterior nerve root (e.g. tumour), (iii) nerve (e.g. peripheral neuritis), (iv) posterior root ganglion (e.g. herpes zoster), (v) posterior root entry zone (e.g. tabes), (vi) muscle (myopathy).

**Site and nature of lesion** These may be obvious through a study of reflexes. For instance, when the knee jerks are lost but the ankle jerks exaggerated, the lesion is likely to be in the region of the third and fourth lumbar segments.

**LATENT REFLEXES** A pyramidal lesion may besides causing exaggeration of deep reflexes bring to the fore some latent reflexes which suggest a pyramidal lesion if present on one side only.

*Hoffman's reflex* (Finger flexion reflex) The examiner supports the patient's hand with the fingers partially flexed. The middle finger is partially extended and either its middle or distal phalanx is grasped firmly between the examiner's index and middle fingers. With a sharp forcible flick of his thumb, the examiner snaps the nail of the patient's middle finger, causing a forcible increased flexion of his finger followed by sudden release. If the reflex is positive there is flexion of the other fingers, including the thumb.

*Wartenberg's sign* With his left hand the examiner tightly grasps the patient's supinated hand at the wrist from below. With his right hand he hooks his four bent fingers into the four bent fingers of the patient. The patient is asked to bend his four fingers further against those of the examiner and to exert all his strength. The examiner pulls against the patient's fingers. When slightest spastic paralysis of the hand exists, the patient's thumb is adducted, flexed and moved inward across the palm. In the normal individual the thumb remains immobile or its end phalanx bends very little.

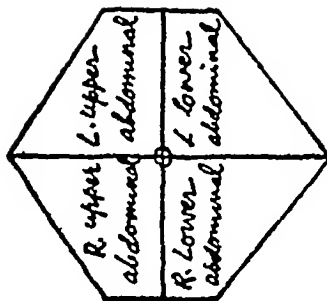
A neurologic reflex chart (Fig. 13.95) is useful for recording the various responses obtained.

**Clonus** A rhythmic, repetitive contraction of a muscle, stimulated through the mechanism of the "stretch reflex" is called a clonus. In the presence of a hyperactive deep reflex, if constant pressure is maintained with a view to keeping the muscle being tested under tension, the reflex may occur repetitively, or keep on recurring. In the great majority of cases, a sustained clonus with exaggerated deep reflexes indicates organic disease of the nervous system.

**ANKLE CLONUS** The extended leg, with the knee slightly bent, is held by the examiner in one hand whilst the foot is grasped with the other. The foot is then briskly dorsiflexed one or more times. A clonic repetition of the ankle jerk or a peculiar rhythmic contraction of calf muscles will be observed in case of a clonus. If the jerking keeps on recurring for as long as the pressure is maintained on the sole of the foot, it is referred to as *true* or *sustained* clonus. If after a few jerks the phenomenon dies out in spite of pressure being maintained, the clonus is called a *false* or *abortive* clonus. False clonus may be observed in normal subjects with hyperactive reflexes and in cases of neurasthenia.

**PATELLAR CLONUS** (Knee clonus) This is elicited by extending the leg, grasping the patella between the thumb and the forefinger and suddenly pushing it down. Clonic contractions of the quadriceps muscle are observed.

	Supinator Jerk	Biceps Jerk	Triceps Jerk	Knee Jerk	Ankle Jerk	Plantar reflex
Right						
Left						



Deep Reflexes      graded    0 - ++++

Plantar    ↓ flexor  
              ↑ extensor  
              ↓ equivocal  
              0 absent

Abdominals    + present  
                      0 absent

Fig 13 95    Method of recording important reflexes



# CLINICAL REFLEXES OF IMPORTANCE (SUMMARY)

Reflex	Segmental innervation	Afferent pathway	Efferent pathway	Required stimulus	Response
<i>Superficial Reflexes</i>					
1 Corneal	Pons	Cr N V	Cr N VII	Touching the cornea	Blinking of eye
2 Conjunctival	Pons	Cr N V	Cr N VII	Touching the conjunctiva	Blinking of eye
3 Pharyngeal	Medulla	Cr N IX	Cr N X	Tickling the pharynx	Gagging or retching
4 Palatal	Medulla	Cr N IX	Cr N X	Tickling the soft palate or uvula	Raising of uvula
5a Abdominal (Epigastric)	T 6, 7, 8, 9	T 6, 7, 8, 9	T 6, 7, 8, 9	Stroking of skin on one side	Muscle contraction
5b Abdominal (mid-abdominal)	T 9, 10, 11	T 9, 10, 11	T 9, 10, 11	Stroking of skin on one side	Muscle contraction with pulling of umbilicus
5c Abdominal (hypogastric)	T11, L2, L1	T 11, L2, L1	T 11, L2, L1	Stroking of skin on one side	Muscle contraction
6 Cremasteric	L 1, 2	Femoral N	Genito femoral N	Stroking of inner surface of thigh	Raising of testicle
7 Gluteal	S 4, 5	Pudendal N	Pudendal N	Stroking of skin over buttock	Contraction of glutei muscles
8 Plantar	L 5, S 1, 2	Tibial N	Tibial N	Stroking of the sole of the foot	Plantar flexion of toes
<i>Deep Reflexes</i>					
1 Jaw	Pons	Cr N V	Cr N V	Tapping the chin	Sudden closure of jaw
2 Pectoral	C5, 6, 7, T1	Pectoral N	Pectoral N	Tapping pectoralis tendon	Contraction of muscle
3 Biceps	C 5, 6	Musculo-cutaneous N	Musculo-cutaneous N	Striking biceps tendon	Flexion of elbow
4 Brachioradial	C 5, 6	Radial N	Radial N	Striking radial styloid process	Flexion and supination of forearm
5 Triceps	C 6, 7, 8	Radial N	Radial N	Striking triceps tendon	Extension of elbow
6 Knee or patellar	L 2, 3, 4	Femoral N	Femoral N	Striking patellar tendon	Extension of knee
7 Ankle or Achilles	L 5, S 1, 2	Tibial N	Femoral N	Striking Achilles tendon	Plantarflexion of foot

## VISCERAL REFLEXES

### Micturition

**Anatomy** **THE BLADDER** The wall of the bladder contains unstriated muscle fibres, which constitute the detrusor muscle. The outlet of the bladder to the urethra is controlled by two sphincters, the internal sphincter which like the detrusor contains unstriated or involuntary muscle fibres, and the external sphincter which consists of striated or voluntary muscle.

**NERVE SUPPLY** The bladder receives its nerve supply from the parasympathetic, which transmits impulses of an involuntary reflex nature, and the cerebro-pudendal pathways, which carry fibres responsible for voluntary control.

The *parasympathetic* supplies the detrusors and sphincters. The pre-ganglionic fibres arise from second, third, and fourth sacral segments and end in the vesical plexus. From there post-ganglionic fibres supply the detrusor and internal sphincter via the pelvic nerves. The external sphincter is supplied by pudic nerves. Stimulation of the parasympathetic results in contraction of the detrusor and relaxation of the internal sphincter, resulting in evacuation of the bladder.

The *cerebro-pudendal pathways* The upper motor neurons after arising from the paracentral lobules, reach the lower part of the spinal cord where the fibres synapse around the anterior horn cells of S2, S3 and S4. The lower motor neurons pass peripherally to supply the external sphincter and carry impulses which bring about voluntary cessation of micturition.

The bladder also receives sympathetic fibres from the upper lumbar segments of the cord, through the hypogastric nerves and ganglia, but these are of little importance.

**NORMAL MICTURITION** As urine accumulates in an empty bladder, the extra volume is accommodated by relaxation of detrusor muscle without allowing intravesical pressure to rise. When urine volume exceeds 100 to 150 ml intravesical pressure increases and the sensation of full bladder intrudes on consciousness, with further distension of bladder, the sensation becomes more and more unpleasant and painful. Despite these sensations voluntary control over the sphincters can be maintained by an adult till the urine volume reaches approximately 450-500 ml.

In the initiation of micturition, the first step is removal of voluntary inhibition. When this occurs, the internal sphincter relaxes, the sacral reflex arc is facilitated, the detrusor contracts, the external sphincter relaxes and bladder is evacuated. During the act, micturition can be voluntarily stopped by contraction of the external sphincter.

### DEFINITION OF BLADDER SYMPTOMS

*Hesitancy* A delay or difficulty in starting urine.

*Urgency or precipitancy* Difficulty in holding urine. The contractions of the wall of the bladder become so vigorous that they cannot be resisted by the external sphincter and micturition becomes precipitate.

*Retention* Impairment of voluntary control of bladder with the result that urine cannot be evacuated at will.

*Dribbling* A type of incontinence in which a few drops of urine keep on escaping from the overfilled bladder.

*Incontinence* (i) Stress incontinence Loss of urine may occur during laughing, coughing, lifting or walking up or down the stairs (ii) Psychologic incontinence The patient retains the desire to void and has the ability to stop micturition but he does not care to stop it and he may void on the floor This happens in the lobotomized patient (iii) Overflow incontinence (iv) Reflex incontinence or automatic bladder (v) Incontinence of non-resistance or dribbling

### Disorders of bladder function

(1) SENSORY DENERVATED BLADDER A lesion selectively affecting the sensory autonomic pathways of the bladder is classically seen in tabes dorsalis Other causes are diabetic autonomic neuropathy, multiple sclerosis, subacute combined degeneration In cauda equina lesions, lesions of sensory roots usually also involve motor roots There is absence of sensation of filling of the bladder, and passage of urine through urethra However as the efferent pathways are intact, patient can be trained to evacuate the bladder periodically If bladder is not evacuated in time, there is overflow incontinence Characteristically patient becomes aware of incontinence only when the clothes become wet.

(2) MOTOR PARALYTIC BLADDER This results from lesions of sacral segments or motor roots The sensations of bladder filling are preserved. Partial lesions produce hesitancy, straining, poor stream and incomplete evacuation Total paralysis causes paralytic atonic bladder with retention and overflow incontinence Pressure on abdominal wall can help in evacuation Infection occurs due to stasis of urine

(3) COMBINED SENSORIMOTOR LESION Lesions of cauda equina, or destructive lesions of sacral spinal segments produce combination of sensory and motor lesions The result is a paralytic, atonic bladder, devoid of sensations, with overflow incontinence Sometimes, the vesical plexus takes over the function and bladder contraction may occur due to local reflex arc Such a bladder is called "autonomous" bladder

(4) TRANSVERSE LESION OF SPINAL CORD ABOVE THE CONUS MEDULLARIS The immediate effects are as follows

(a) *Retention of urine* The detrusor becomes toneless and fails to respond, by contraction, even when stretched by accumulated urine, the internal sphincter remaining contracted

(b) *Overflow incontinence* When the bladder cannot be further distended, the intravesical pressure may force open the internal sphincter, resulting in dribbling incontinence As the external sphincter is also toneless, voluntary control is not possible

(c) *Automatic micturition* (i) In the absence of secondary infection of the bladder, after the stage of shock has passed off, the detrusor regains its tone

When its tone reaches a certain intensity, the contraction of the internal sphincter is overcome and urine escapes. Reflex or automatic micturition, although usually independent of external stimuli, may be initiated by reflex stimuli. In slowly progressive or partial damage to the cord, the usual sequence is difficult micturition or there is delay in starting micturition (or a weak stream) followed by retention of urine and, later, dribbling incontinence and automatic micturition ("automatic" bladder).

(ii) Infection of the bladder causes failure of hypertrophy of the detrusor and bladder automaticity does not develop. Either over-distension with overflow incontinence persists or the condition reverts back to one of retention of urine.

(5) BILATERAL LESIONS OF THE PARA CENTRAL LOBULES OR INTERNAL CAPSULES. This may be due to superior longitudinal sinus thrombosis which is of rare occurrence. This condition is characterized by a sequence of three stages namely retention, overflow incontinence and automaticity. In advanced cases, the bladder tonus may become so great, that even a small amount of urine may initiate micturition.

### Defaecation

**Anatomy.** The nerve supply of the rectum is similar to that of the bladder. The parasympathetic centre is situated in segments S 3, 4 and 5. Parasympathetic impulses induce contraction of sigmoid and rectum and relaxation of internal sphincter. Afferent impulses about degree of rectal filling are carried to the cerebrum mainly by parasympathetic and to some extent by sympathetic fibres. Efferent voluntary impulses from the cerebrum are carried by fibres which descend in the lateral columns and reach the anterior horn cells of Th 6 to Th 12, which are concerned with contraction of the abdominal muscles which constitute an important detrusor of the rectum.

**Disturbances of rectal function.** *Transverse lesions of spinal cord above sacral level.* There is faecal retention due to loss of voluntary control of sphincter ani and sensation of rectal urgency. Occasionally, there is reflex intermittent involuntary defaecation.

*Lesions of sacral cord.* Faecal incontinence results due to paralysis of sphincter ani. Hard faecal masses may be retained.

### DISTURBANCE OF SEXUAL FUNCTION

**Impotence** is a relative or complete inability to initiate, sustain or successfully conclude the act of sexual intercourse. A majority of cases of impotence are psychogenic in origin. Impotence can be due to structural damage to the lumbo-sacral plexus, cauda equina or conus medullaris. Neurological conditions with which impotence may be associated include tabes dorsalis, taboparesis or other varieties of neuro-vascular syphilis affecting the cord, disseminated sclerosis and some cases of progressive muscular atrophy as well as all traumatic lesions affecting the spinal cord at or above the level of the first lumbar vertebra. Diabetic autonomic neuropathy is another important cause.

# INNERVATION OF THE BLADDER AND URETHRA IN MAN

<i>Fibres</i>	<i>Segment</i>	<i>Peripheral nerves</i>	<i>Structure innervated</i>	<i>Function</i>
		Efferent		
Sympathetic	L 1, 2, 3	Sup hypogastric plexus— Presacral and hypogastric nerves	Detrusor Int sphincter	Inhibitory Facilitatory
Parasympathetic	S 2-4	Pudendal plexus— pelvic nerves	Detrusor Int sphincter Ext sphincter	Facilitatory Inhibitory Motor
Somatic voluntary	S 2-4	Pudendal plexus— pudendal nerve		
		Afferent		
Sympathetic	Detrusor	Same as efferent	Cells in intermediolateral column of cord L2-3	Pain
Parasympathetic	Detrusor Int sphincter Ext sphincter Post urethra	Same as efferent	S 2-4 grey matter	Stretch pain
Somatic sensory		Same as efferent	S 2-4	Temperature Pain

SUPRASPINAL HIGHER CONTROL			
<i>Fibres</i>	<i>Course</i>	<i>"Centre"</i>	<i>Function</i>
Afferent	Antero-lateral column of sp cord	Hypothalamus Post central gyrus	Pain Fullness
Efferent	Mid lateral column of sp cord	Hypothalamus Precentral gyrus Frontal lobe	Voluntary motor control



Fig 1396 Charcot's joint Arthropathy of the left knee joint with subluxation in a case of tabes dorsalis

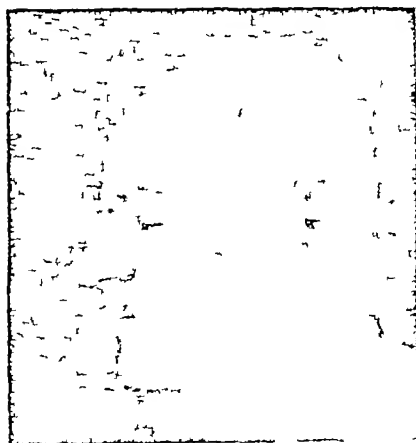


Fig 1397 Deformity of the skull due to intracranial neoplasm (meningioma)

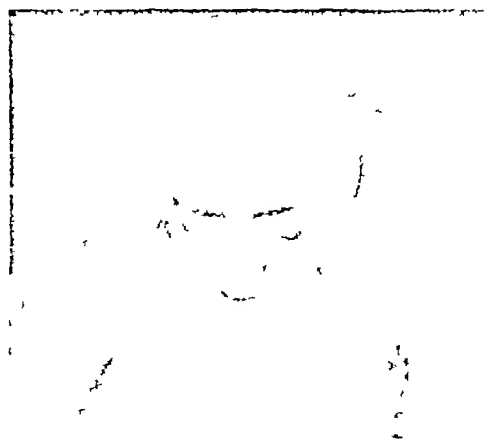


Fig 1398 Secondary deposits in skull from acute lymphoblastic leukaemia



## TROPHIC AND VASOMOTOR CHANGES

Nutritional or trophic changes form an important part of examination in many cases of neurological disease. Each nerve cell constitutes the trophic centre for its fibrés, the trophic centres in the case of motor nerves being located in anterior horn cells, those of sensory nerves in the posterior root ganglion cells. Trophic disturbances of neurological origin may manifest themselves in the skin, nails, subcutaneous tissues, muscles, bones or joints.

**Skin.** The skin may become thickened (hyperkeratosis), pigmented and glossy, display alterations of colour, patchy pigmentation, cutaneous ecchymoses or even cyanosis. There may be loss of sweating or excessive sweating, particularly over the face and upper limbs. The commonest trophic lesion in case of tabes is a perforating ulcer, either underneath the pad of the great toe or at any pressure-point on the sole of the foot (Fig 13 96). Decubital ulcers and ulcers of the tips of the fingers may also arise. As part of the trophic disturbance, loss of hair may occur, nails may become thick, curved and brittle.

**Joints.** Joints may display arthropathy, with painless swelling, effusion and oedema. The knee joint is most commonly affected (Fig 13 97), less often the hip, shoulder, tarsal joints, elbow, ankle and small joints of fingers and toes. There is proliferation and destruction of cartilage and bony surfaces and formation of new bone fragments some of which may be free within the joints.

**Bones.** Fragility of bones, decalcification and fractures may occur. Leprosy, syringomyelia and tabes dorsalis are the diseases in which trophic disturbances are commonly observed.

## CRANIUM AND SPINE

Some neurologists prefer to begin with the spine and cranium in an examination of the nervous system. The bony cage of the central nervous system may reflect diseases of this system or vice versa.

**Cranium** (1) Size and shape of head. Any macrocephaly or microcephaly? (2) Retraction of head in meningitis and torticollis. (3) Deformity, such as oxycephaly. Tenderness on palpation or percussion may be found in the region overlying a tumour. A bony boss may overlie a meningioma (Fig 13 98). Deposits in the skull may occur in leukaemia and malignancy (Fig 13 99). (4) Percussion of the skull may disclose dullness over a subdural haematoma. Separation of sutures due to hydrocephalus may yield a cracked-pot sound. (5) Bulging of fontanelles in children suggests increased intracranial pressure. (6) Congestion and tortuosity of the arteries of the scalp may sometimes be associated with a vascular intracranial tumour. There may be thickening and tenderness of the temporal arteries in temporal arteritis. (7) Auscultation. In vascular anomalies, like angioma, and less frequently vascular tumours



and intracranial aneurysms, auscultation of the skull over the eyeballs or the site of lesion may reveal a bruit. It should be noted if the bruit alters in character on compression of the carotid artery or jugular vein on either side.

**Spine** The spine is examined for deformity, rigidity and tenderness. One or more of these may be found in diseases leading to spinal compression.

(1) *Kyphoscoliosis* Such abnormality of spinal curvature may be congenital, idiopathic or associated with neurological disease such as Friedreich's ataxia, neurofibromatosis, syringomyelia, cerebral palsy or poliomyelitis.

(2) *Lordosis* of marked degree causing an arched back is seen with muscular dystrophies and myopathies.

(3) *Kyphosis or gibbus* is common in tuberculosis of the spine, not an uncommon cause of paraplegia.

(4) *Rigid spine* Rigidity of the spine is common in spinal tuberculosis. In protruded disc of cervical spondylosis the mobility of the cervical spine tends to be restricted. On forced flexion of the neck, the feeling of an electric current passing down the vertebral column, and often into the arms and legs may be present (Lhermitte's sign, barber chair sign). The same phenomenon may occur with other causes of cervical cord compression such as arachnoiditis, space occupying lesions, etc. In case of lumbar spondylosis there is restriction of spinal movements at that level and the spinal muscles are tense. In both cervical and lumbar lesions there may be an increase in local pain and pain of radicular distribution on movement of the spine, and on coughing, sneezing or straining.

(5) *Tender spine* Local areas of tenderness may be elicited by gentle percussion of the spine. Tenderness can occur with tuberculosis or other infection, tumors of the vertebra or secondary carcinomatous deposits, syphilitic osteitis and osteitis deformans, apart from traumatic injury.

## STANCE

The patient is asked to stand erect as still as possible. His posture and presence of involuntary movements, if any, are observed. The patient is then asked to stand erect with his feet together and eyes open. Any tendency to sway or lose balance should be noted. Remaining in this posture, the patient is next asked to close his eyes, care being taken to see that he does not fall.

### ABNORMALITIES OF STANCE

1 *Akinesia* In Parkinsonism the patient stands slightly stooped, the face is expressionless, and the head is turned in company with the shoulders and trunk.

2 *Abnormalities of equilibrium* (1) *Swaying* When a normal individual stands with his feet together, there is a tendency to sway slightly. In cerebellar disease the swaying is markedly accentuated and the patient may sway wildly from side to side but will not fall. Inability to remain upright in absence of paralysis, may also be met with in vertebrobasilar ischemia due to

atherosclerosis, or vestibular lesion on the affected side, or may be hysterical (b) *Tendency to fall backwards* may be encountered in patients with basilar atherosclerosis, lesions of the cerebellar vermis, or of foramen magnum e.g. Arnold-Chiari malformation, the tendency to fall backwards being especially so on looking upwards (c) *Standing on a wide base* with feet well apart is seen either with dorsal column lesion, or cerebellar disease, the differentiation can be made by Romberg's test

3 *Squatting and standing up* Due to weakness of proximal leg and paraspinal muscles, the patient rises by 'climbing up his legs' in muscular dystrophies, (Fig 13.99) some cases of myasthenia, polymyositis, and chronic polyneuropathies

## GAIT

In the course of evolution, the pronograde or four-legged gait of animals has been replaced in man by the plantigrade or two-legged gait, because of his adoption of the upright posture for standing

A normal gait may become disordered as the result of loss of muscle power, alteration of muscle tone, incoordination of movement, deformity, pain during movement or hysteria. The majority of abnormal gaits are due to disease of the nerve system or of the muscles

To observe the gait ask the patient to walk normally and follow him as he walks, being ready to support him should he tend to fall. Where there is a minimal disorder of gait, more elaborate tests may be employed such as heel-to-toe walking (tandem walking), or hopping on one or alternate legs. The patient may also be asked to walk forward, then turn round quickly and then walk along a straight line for about 30 feet (approximately 10 metres), then turn and go back to the starting point

The following types of abnormal gait are particularly noteworthy, with familiarity and practice, most of them are easily identifiable

Hemiplegic gait (Circumduction gait) This is a variety of "spastic" gait commonly encountered in cases of hemiplegia and indicative of a unilateral pyramidal or upper motor neurone lesion, resulting in unilateral spasticity of muscles. At each step, the pelvis is tilted sideways, the affected and rigid lower limb is stiffly dragged sideways and forwards in semi-circular fashion, the inner side of the foot scraping the floor, the paralytic arm is usually kept flexed and immobile

Spastic gait (Sticky gait) This is another variety of gait, encountered in cases of spastic paraplegia or diplegia, and indicative of bilateral pyramidal disease, as in subacute combined degeneration, disseminated sclerosis, spinal

compression and syringomyelia The patient's legs are held stiffly together, the pelvis being tilted from side to side and the toes scraping the floor on either side

Scissors gait (Cross-legged gait) An exaggerated form of paraplegic gait, with excessive spasticity of the adductor muscles of the thighs, resulting in the legs being either closely approximated or crossing each other on walking, as in cases of Little's disease or cerebral diplegia in children

The term *spastic gait*, indicative of increased muscle tone, has been applied indiscriminately to a variety of gaits, such as the hemiplegic, paraplegic and scissors gait, and is therefore best avoided

Shuffling gait (Parkinsonian gait, festinant gait, festinating gait) The patient, with the head and trunk bent forwards and the limbs partially flexed, walks with short, rapid shuffling steps There is loss of swinging of the arms when walking When pushed forwards (propulsion), backwards (retropulsion) or sideways (lateripulsion), he is unable to pull himself up, the walk frequently developing into a trot, shuffle or fall, he gives the appearance of "chasing his centre of gravity" This type of gait is encountered in Parkinson's disease, cerebral arteriosclerosis, and hepatolenticular degeneration

Ataxic gait (Reeling gait, staggering gait) (1) CEREBELLAR ATAXIC GAIT Encountered in cerebellar disease, disseminated sclerosis, Friedreich's ataxia and hereditary cerebellar ataxia, this type of gait is characteristic The feet are planted wide apart and the patient walks in a swaying, reeling, staggering or uncertain manner like a "drunk", and with a tendency to swing to one or other side The gait of a person with a disturbance of the vestibular apparatus may also be reeling The tendency to walk towards the side of lesion and in a circle (compass gait), though characteristic of unilateral cerebellar lesion, may be encountered in unilateral labyrinthine disorder

(2) SENSORY ATAXIC GAIT (Tabetic gait, stamping gait) This type of gait may be noted in tabes dorsalis, polyneuritis, cauda equina lesion or subacute combined degeneration The patient walks with the feet wide apart, the leg is suddenly raised high, thrown forward or waved about in an uncertain manner and then brought down, heel first, in a noisy or stamping manner His eyes are firmly glued to the ground whilst walking, as observed by Osler, many years ago, "the normal man walks by faith, the tabetic by sight"

The cause of an ataxic gait cannot regularly be discovered from observation of the gait alone

High-steppage gait (Flaccid gait, prancing gait) In polyneuritis, polio-myelitis, cauda equina tumour or in external popliteal nerve lesion, because of foot-drop, the foot is raised abnormally high in order to clear the ground and is then brought down with a slap

**Jaunty gait** (Dancing gait) Due to a combination of jerky involuntary movements with muscular hypotonia, the gait in chorea displays a characteristic "jauntiness" and unpredictability of behaviour. In the patient of multiple sclerosis, due to a combination of spasticity, and ataxia, the weight bearing extremity may display dancing or bounding movements which are rapidly repeated, giving rise to irregular, up-and-down movement of the entire body.

**Hysterical gait** There is no standard or "set" pattern for this gait. The gait in hysteria is usually inconsistent, bizarre, associated with emotional upheavals, displays no signs of organic neurological involvement, does not conform to any recognized type of gait and becomes particularly noticeable in the presence of bystanders.

**Waddling gait** (Oscillating gait) The body sways or waddles from side to side like that of a duck, the feet are planted wide apart, the shoulders thrown back and the abdomen pushed forwards. This type of gait may be due to congenital dislocation of the hips, muscular dystrophy or myopathy, massive abdominal tumour, ascites or late pregnancy.

In the patient with muscular dystrophy, weakness of the extensors of the spine and knees leads to the adoption of a characteristic method of standing up from the ground—"climbing or walking up his own legs". The boy rolls over on his abdomen, then slowly gets on to his hands and knees, keeps his hand on the floor as long as possible and then rises by claspings his legs and knees alternately until he gradually brings his body into the erect position (Fig 13 99).

In advanced stages of muscular dystrophy, the patient may not be able even to stand erect but may be able to move about on his fingers and toes in a peculiar frog-like gait.

**Myotonic gait** In dystrophia myotonica or myotonia congenita, failure of the muscles to relax properly after contraction results in a peculiar gait interrupted by severe tonic spasms of muscles. Any attempt to walk, turn or change the speed of walking, results in a prolonged spasm or myotonus with a transitory incapacity to walk.

**Limping gait** This may be due to pain from fracture, arthritis, sprain or painful callosity, or a deformity, such as congenital dislocation of the hip, club foot, or unequal length of the legs. The limp may be either painful or painless, in the former type, the patient avoids putting his weight on the affected leg whilst walking.

**Kinesia paradoxa** Sometimes observed in Parkinsonism, kinesia paradoxa represents ability to run better than walk.

**Tandem walking** Patient is made to walk heel-to-toe in a straight line. It is a good test for the integrity of the vermis of the cerebellum which may be affected in alcoholic cerebellar degeneration.

**Apraxia of gait** The patient seems to have forgotten how to use the muscles to walk and the steps are small and uncertain. He often stops and has to be plodded on to move. This type of gait may be observed in lesions of the frontal lobe or bilateral damage to cortico-spinal tracts in internal capsule, central peduncles or high brain stem e.g. pseudo bulbar palsy

**Gait in intermittent claudication** The patient walks normally at first, but after going some distance, he stands still or sits down to rest till the spasm relaxes and then starts off again. This is usually due to defective blood supply to the leg muscles, secondary to peripheral vascular disease.

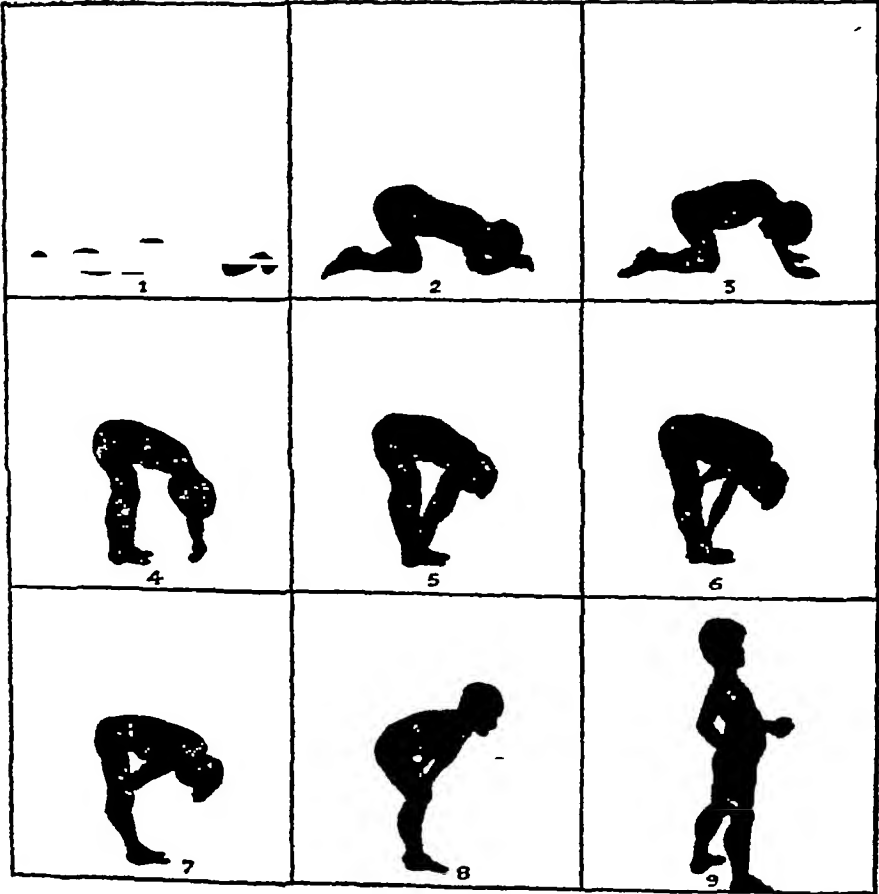


Fig 13.99 Method of rising from the supine to the erect position in pseudo hypertrophic muscular dystrophy

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To be read with page 297

## CONTINUOUS MURMURS

(3) *Austin Flint murmur* A mid-diastolic or presystolic murmur almost indistinguishable clinically from that of mitral stenosis may be heard at the apex in a case of severe aortic regurgitation. A clear cut history of pneumonic fever in the past the presence of an accentuated first sound or opening snap and a third sound over the left ventricle may however suggest organic mitral stenosis. The use of amyl nitrite may help to identify the murmur of mitral stenosis, for the rumble of a stenotic valve will accentuate with the postamyl tachycardia, while the Austin Flint rumble will decrease in response to the lowered peripheral resistance and blood pressure (and associated reduction in the degree of aortic regurgitation). Also a mid-diastolic or presystolic murmur at the apex cannot be due to mitral stenosis if the aortic incompetence is syphilitic in origin. Mechanism The Austin Flint murmur has been attributed to a variety of mechanisms including the vibrations set up in the antegrade stream of blood from the left atrium and retrograde stream of blood from the aorta. However the most likely explanation is that it is the result of forward flow across a closing mitral valve (functional mitral stenosis) which may result from abnormal ventricular filling or from the aortic regurgitation jet impinging on the leaflet during diastole.

